



Delayed Neurotoxic Envenomation Following Yellow Cobra Bite Leading to Acute Respiratory Failure: A Case Report

Yulian Ponco Wibowo¹, Yusti Desita Indriani²

¹ Faculty of Medicine, Universitas Islam Sumatera Utara, Indonesia

² Faculty of Medicine, Universitas Sriwijaya, Indonesia

Corresponding Email : poncowibowo10@gmail.com

Article History :

Received date : 2026/04/17

Revised date : 2026/05/05

Accepted date : 2026/06/21

Published date : 2026/07/14



Copyright: © 2024 by the authors. Submitted for possible open access publication under the terms and conditions of the Creative Commons Attribution (BY NC) license (<https://creativecommons.org/licenses/by-nc/4.0/>).

E-ISSN :

ISSN 3048-1368



P-ISSN

ISSN 3048-1376



ABSTRACT

Introduction: Neurotoxic envenomation by *Naja sumatrana* (yellow cobra) represents a life-threatening medical emergency in Southeast Asia. Delayed hospital presentation significantly aggravates the clinical course and may culminate in acute respiratory failure requiring intensive care. We report a case of severe cobra envenomation with an 8-hour pre-hospital delay leading to progressive neurotoxicity and acute respiratory failure mandating mechanical ventilation.

Case Illustration: A 21-year-old male agricultural worker presented to Rumah Sakit AR. Bunda Lubuklinggau, South Sumatera, 8 hours after sustaining a yellow cobra bite to the right cruris while tapping rubber in a plantation. On arrival, the patient was somnolent with a deteriorating Glasgow Coma Scale (GCS) of E3M6V4, tachycardia (HR 104 bpm), tachypnoea (RR 23 breaths/min), and SpO₂ 98% on supplemental oxygen. Local

examination revealed puncture marks, pain, and oedema at the bite site. Laboratory investigation disclosed leukocytosis (16,200/ μ L), elevated erythrocyte sedimentation rate (ESR; 21 mm/hour), and eosinophilia (7%). The patient was classified as snakebite Grade III with impending respiratory failure. Despite initial anti-snake venom (ASV) administration of 2 vials, neurological deterioration progressed requiring emergency orotracheal intubation and admission to the Intensive Care Unit (ICU). A total cumulative ASV dose of 20 vials was administered over 24 hours per anesthesiologist protocol, in addition to broad-spectrum antibiotic therapy (meropenem 3 $\times$ 1 g), ketorolac, and central venous catheterisation. The patient achieved satisfactory clinical improvement and was successfully weaned from mechanical ventilation.

Discussion: This case highlights the critical impact of delayed medical presentation on the severity of neurotoxic cobra envenomation. Alpha-neurotoxins of *Naja sumatrana* competitively and reversibly block post-synaptic nicotinic acetylcholine receptors, producing descending flaccid paralysis that may progress to acute respiratory failure if not promptly treated. Delayed antivenom administration, compounded by initial subtherapeutic dosing due to limited vial availability, resulted in progressive envenomation requiring mechanical ventilation and high-dose repeated ASV therapy. This case underscores the importance of early recognition, rapid referral, adequate antivenom supply, and multidisciplinary ICU management in rural healthcare settings.

Conclusion: Delayed hospital presentation following neurotoxic cobra bite is associated with life-threatening respiratory failure. Early identification of neurotoxic signs, prompt emergency

referral, aggressive antivenom administration, and airway protection are paramount to favourable outcomes. Rural healthcare facilities must maintain adequate ASV stockpiles and capacity for emergency intubation.

Keywords: Naja sumatrana; neurotoxic envenomation; acute respiratory failure; anti-snake venom; mechanical ventilation

INTRODUCTION

Snakebite envenomation constitutes a major neglected tropical disease affecting an estimated 1.8 to 2.7 million individuals annually, resulting in 80,000 to 138,000 deaths and over 400,000 cases of permanent disability worldwide.^{1,2} The World Health Organization (WHO) elevated snakebite envenomation to Category A status within the Neglected Tropical Diseases list in 2017, reflecting its disproportionate burden on impoverished rural populations in the tropics.²

Southeast Asia harbours a diverse spectrum of medically significant venomous elapids, among which *Naja sumatrana* (Equatorial Spitting Cobra, commonly identified by its distinctive yellow coloration in juvenile and sub-adult forms) is an important cause of morbidity and mortality in Indonesia, Malaysia, and the surrounding region.³ In Malaysia, *N. sumatrana* bites accounted for 28.1% of confirmed *Naja* species envenomations over a four-year period, with systemic manifestations including ptosis, vomiting, and local oedema documented in a substantial proportion of victims.⁴

The pathophysiological basis of neurotoxic cobra envenomation resides in the action of three-finger α -neurotoxins (3FTx), which competitively block post-synaptic nicotinic acetylcholine receptors (nAChR) at the neuromuscular junction, producing a dose-dependent descending flaccid paralysis that may culminate in acute respiratory failure.^{5,6} The pharmacokinetics of *N. sumatrana* venom demonstrate rapid absorption of the principal neurotoxin following intramuscular injection (Tmax approximately 30 minutes), with high intramuscular bioavailability (81.5%), facilitating swift systemic toxicity.⁷

Delayed presentation to medical facilities is a critical determinant of clinical outcome in neurotoxic envenomation. Systematic evidence indicates that a bite-to-care interval exceeding 6–12 hours is strongly associated with severe systemic toxicity, including respiratory failure and death.^{8,9} Furthermore, time from snakebite to antivenom administration directly correlates with the severity and duration of neuromuscular paralysis.¹⁰

This case describes a patient bitten by a yellow cobra who arrived at a secondary rural hospital 8 hours post-envenomation, demonstrating progressive neurotoxicity culminating in acute respiratory failure requiring mechanical ventilation and high-dose cumulative antivenom therapy.

Research Objective: To document the clinical trajectory, diagnostic assessment, therapeutic management, and multidisciplinary ICU outcomes of delayed neurotoxic yellow cobra envenomation progressing to acute respiratory failure.

Research Benefit: This case report provides practical clinical insights for emergency physicians, intensivists, and rural healthcare practitioners on the management of severe neurotoxic cobra envenomation with delayed presentation, and highlights systemic deficiencies in pre-hospital snakebite first aid and antivenom availability.

Research Gap: Existing literature predominantly addresses early-presentation cobra envenomation. Cases specifically documenting the clinical consequences and therapeutic requirements of 8-hour delayed presentation of *N. sumatrana* envenomation with resultant acute respiratory failure requiring mechanical ventilation and repeated ASV dosing remain underrepresented in peer-reviewed literature from Indonesia.

Novelty: This is the first documented case report from a secondary hospital in Lubuklinggau, South Sumatera, describing the comprehensive emergency and ICU management trajectory of delayed *N. sumatrana* (yellow cobra) envenomation-induced acute respiratory failure, with explicit documentation of high-dose cumulative antivenom protocol and outcomes.

CASE ILLUSTRATION

Patient Identification and Background

The patient is an AN. S., a 21-year-old male (date of birth: 09 October 2004), No. RM 26015077, with blood group O-positive, presenting without history of prior systemic illness or known drug allergy. He works as a rubber tapper (penyadap karet) in a plantation area in the Lubuklinggau region, South Sumatera, Indonesia. His health insurance is registered as PRIBADI/UMUM.

Chief Complaint and History of Present Illness

The patient was referred to the Emergency Department (IGD) of Rumah Sakit AR. Bunda Lubuklinggau on 8 July 2026 at 19:49 local time with a chief complaint of progressive somnolence, chest discomfort, and generalised weakness.

Eight hours prior to hospital arrival (approximately 11:49 local time), the patient was bitten on the right lower leg (cruris dextra) by a yellow-coloured cobra while conducting rubber tapping activities in a remote plantation area. The patient identified the offending snake as a yellow cobra (ular kobra berwarna kuning), consistent with *Naja sumatrana* based on regional distribution and characteristic coloration. No first aid such as immobilisation bandaging or tourniquet application was documented.

Following the bite, the patient reported: (1) progressive general malaise and profound weakness (lemas); (2) four episodes of vomiting (emesis 4×) consisting of ingested food contents; (3) a generalised sensation of body coldness (badan pasien dingin); and (4) headache (nyeri kepala). Seizure activity (kejang) and loss of consciousness (pingsan) were explicitly denied. Chest pain (nyeri dada) was noted to have developed in the period prior to hospital arrival.

The patient was transported to the hospital without prior medical attention at any intermediate health facility, resulting in a total pre-hospital interval of 8 hours from the time of envenomation to first physician contact.

Physical Examination on Arrival

General Status: The patient appeared somnolent and distressed. Nutritional and hydration status appeared mildly impaired. Verbal contact was obtainable but limited.

Vital Signs:

- Consciousness: Composmentis (CM) → deteriorating to E3M6V4 (GCS 13)
- Blood pressure: 144/52 mmHg (initial) → 111/70 mmHg (after fluid resuscitation)
- Heart rate: 104 bpm (regular)
- Respiratory rate: 23 breaths/minute
- Temperature: 36.6°C
- SpO₂: 98% (on supplemental oxygen)
- Visual Analogue Scale (VAS) pain score: 7/10

Head and Face: No conjunctival anaemia (CA -/-), no scleral icterus (SI -/-), nasal and oral cavities within normal limits.

Neck: Within normal limits. No lymphadenopathy or venous distension observed.

Thorax:

Cardiology: Heart sounds S1-S2 regular, no murmur, no gallop detected.

Pulmonology: Symmetrical thoracic expansion bilaterally; percussion sonorous bilaterally; breath sounds vesicular bilaterally (+/+); no rhonchi (-/-); no wheezing (-/-).

Abdomen: Distension observed without fluid wave (undulasi -); no tenderness on palpation; bowel sounds present (+); no flatus; no costovertebral angle (CVA) tenderness.

Extremities: Peripheral pallor noted (akral pucat), indicative of peripheral vasoconstriction or early circulatory compromise.

Skin: No rash, no necrotic lesion, no urticaria.

Neurological Status: Initial neurological examination within normal limits; however, GCS declined to E3M6V4 (total 13) during observation period, indicating progressive encephalopathic deterioration secondary to systemic envenomation.

Locus Status (Right Cruris): Visible fang puncture marks (bekas gigitan) with surrounding localised pain (nyeri) and mild to moderate oedema (bengkak) at the envenomation site. No significant ecchymosis, blister formation, or early necrotic change observed at time of initial examination.

Laboratory Investigations

Complete blood count and biochemical panel obtained on 8 July 2026 at 19:52 (approximately 8 hours post-envenomation). Results from RS AR. Bunda Lubuklinggau, interpreted by dr. M. Taufan Lutfi A., Sp.PK, MARS:

Parameter	Result	Normal Range	Interpretation
Anti-HIV	Non-reactive	Non-reactive	Normal
Glucose (random)	87 mg/dL	74–120 mg/dL	Normal
Sodium (Na ⁺)	139 mEq/L	135–148 mEq/L	Normal

Potassium (K ⁺)	4.1 mEq/L	3.5–5.5 mEq/L	Normal
Chloride (Cl ⁻)	98 mEq/L	95–108 mEq/L	Normal
Blood group	O Rhesus (+)	—	—
Haemoglobin	16.7 g/dL	13.5–17.5 g/dL	Normal
Leukocyte count	16,200 / μ L *	4,500–13,500 / μ L	Elevated $\uparrow$
ESR (BSE) - 1st hour	21 mm/hr *	1–15 mm/hr	Elevated $\uparrow$
Basophil	1%	0–1%	Normal
Eosinophil	7% *	2–4%	Elevated $\uparrow$
Segmented neutrophil	61%	40–70%	Normal
Lymphocyte	25% *	30–45%	Mildly low $\downarrow$
Monocyte	6%	2–10%	Normal
Erythrocyte	5.5 jt/mm ³	4–5.9 jt/mm ³	Normal
Thrombocyte	264,000 / μ L	150,000–450,000 / μ L	Normal
Haematocrit	47%	35–47%	Normal (upper limit)
MCV	86 fL	80–100 fL	Normal
MCH	30 pg	27–32 pg	Normal
MCHC	35 g/dL	32–36 g/dL	Normal
HBsAg	Non-reactive	Non-reactive	Normal
Creatinine	0.94 mg/dL	0.9–1.3 mg/dL	Normal
Ureum	21 mg/dL	19–44 mg/dL	Normal

Abnormal findings are marked with an asterisk (*). Leukocytosis (16,200/ μ L) likely reflects a stress leucocytosis response to systemic envenomation. Eosinophilia (7%) may represent an immune response to foreign venom proteins. Elevated ESR suggests an ongoing inflammatory

response. Renal and hepatic function markers (creatinine and ureum) remain within normal limits, arguing against early nephrotoxicity.

Working Diagnosis

The clinical diagnosis was established as: severe systemic envenomation with threatened respiratory failure, classified as Acute Respiratory Failure (ARF) secondary to neurotoxic elapid envenomation upon ICU admission.

Initial Emergency Management

The following therapeutic interventions were initiated in the Emergency Department upon physician assessment:

1. Intravenous fluid resuscitation: Ringer Lactate (RL) drip at 20 gtt/minute
2. Anti-snake venom (ABU/ASV): 5 vials diluted in Dextrose 5% 100 mL — only 2 vials were immediately available at the facility
3. Ketorolac 1 ampule IV (analgesic)
4. Omeprazole 1 ampule IV (gastrointestinal mucosal protection)
5. Ondansetron 1 ampule IV (antiemetic)
6. Supplemental oxygen: 10 L/min via non-rebreather mask (NRM)
7. Urinary catheterisation (Dower catheter/DC) for strict intake-output monitoring
8. 12-lead electrocardiography (ECG)
9. Urgent anaesthesiology consultation (dr. Sardimon, Sp.An): ICU admission approved

ICU Admission and Clinical Course

Following emergency stabilisation, the patient was transferred to the Intensive Care Unit. On ICU assessment, the patient was placed on mechanical ventilation due to progressive neuromuscular weakness and deteriorating respiratory drive consistent with acute respiratory failure secondary to neurotoxic envenomation.

The attending anaesthesiologist (dr. Sardimon, Sp.An) initiated the following advanced therapeutic protocol in the ICU:

Antivenom Protocol: ABU 8 vials diluted in Dextrose 5% 100 mL — administered over 2 hours. Repeat assessment conducted every 6 hours. If no clinical improvement observed within 6

hours, an additional 2 vials were administered. The total target was a cumulative dose of 20 vials of ASV within 24 hours — in accordance with the anesthesiologist's protocol for severe neurotoxic elapid envenomation.

Mechanical Ventilation: The patient was orally intubated and placed on volume-controlled ventilation via endotracheal tube. Settings were adjusted by the ICU team to maintain adequate oxygenation and prevent ventilator-induced lung injury.

Additional ICU medications: Ketorolac 3×30 mg IV (analgesia), Meropenem 3×1 g IV (broad-spectrum antibiotic prophylaxis for aspiration pneumonia risk and secondary infection prevention).

Central venous catheter (CVC) placement was performed for haemodynamic monitoring, reliable intravenous access, and administration of continuous infusion medications.

Clinical monitoring during ICU stay included: continuous electrocardiographic monitoring via cardiac monitor (Mindray), haemodynamic assessment via automated NIBP (non-invasive blood pressure), syringe pump infusion monitoring (Terumo), and continuous pulse oximetry. Monitoring records from bedside showed HR 77 bpm, SpO₂ 100/minute, RR 22 breaths/minute, blood pressure 263 (systolic reading on monitor scale) corresponding to haemodynamic recovery trajectory.

Progressive clinical improvement was noted on serial assessment, with the patient eventually achieving successful ventilator weaning and extubation following adequate neutralisation of circulating venom and recovery of neuromuscular function.

Clinical Photographs



Figure 1: Patient on mechanical ventilation in the ICU with orotracheal tube in situ, central venous catheter in right internal jugular position, and cardiac monitoring electrodes attached. Bilateral lower limb restraints applied for patient safety.



Figure 2: ICU monitoring array demonstrating Mindray infusion pump (rate 60 mL/hr), syringe pump (Terumo; rate 4.00 mL/hr, 5.65 mL delivered), and bedside cardiac monitor (HR 72, SpO2 100, RR 21).



Figure 3: Close-up view of the right cruris demonstrating two fang puncture marks with surrounding oedema, consistent with elapid envenomation site.



Figure 4: Full-body view of patient in ICU demonstrating ventilator-dependent state with bilateral ankle restraints, cardiac monitoring, NIBP cuff on left upper arm, and nasogastric tube in situ.

DISCUSSION

Identification of the Offending Snake

The patient explicitly identified the offending snake as a yellow cobra encountered in a rubber plantation in Lubuklinggau, South Sumatera, Indonesia. Based on taxonomic distribution and morphological characteristics — specifically the distinctive yellow coloration commonly observed in sub-adult specimens of *Naja sumatrana* (Equatorial Spitting Cobra, formerly classified as *Naja sputatrix*) — the causative species is most consistent with *N. sumatrana* or possibly *N. kaouthia* (Monocled Cobra), both of which are medically significant elapids prevalent in Sumatera and Peninsular Malaysia.^{3,4}

In a comprehensive cohort study of *Naja* species envenomations in Malaysia over 2020–2023, *N. sumatrana* was documented as the second most common offending species (28.1% of cases), with a nationwide distribution. Lower limb bites predominated (52.1%), a pattern consistent with occupational agricultural exposure, as in the present case.⁴ Pharmacokinetic studies on *N. sumatrana* venom have demonstrated that the neurotoxin fraction achieves peak serum concentration within 30 minutes following intramuscular injection, with an intramuscular bioavailability of 81.5%, underscoring the rapid systemic progression of envenomation even without intravenous injection.⁷

Impact of Delayed Hospital Presentation

The most clinically consequential feature of this case is the 8-hour interval between envenomation and hospital arrival. This delay substantially exacerbated the degree of systemic envenomation and ultimately contributed to the development of acute respiratory failure.

A systematic review and meta-analysis of global snakebite morbidity confirmed that snakebite incidents occur most frequently during daytime in agricultural settings, affecting predominantly male rural workers — a pattern precisely mirrored in this case.⁹ The Rational Clinical Examination systematic review demonstrated that a bite-to-care interval of ≥ 6 hours significantly increases the likelihood of severe systemic envenomation (Likelihood Ratio 3.4; 95% CI 1.1–6.4).¹¹

Evidence from Sri Lanka documented that treatment-seeking delay remained common despite proximity to health facilities, with antivenom administration occurring a median of 230 minutes post-bite in a cohort where 78% of transferred patients already manifested systemic envenomation signs on arrival.¹⁰ In the present case, the 8-hour pre-hospital interval allowed near-complete distribution of the absorbed neurotoxin fraction to peripheral tissues and neuromuscular junctions, rendering initial antivenom administration partially less effective than would be expected with early treatment.

A prospective study from Coastal Karnataka demonstrated that patients reaching healthcare facilities within 30 minutes had significantly higher recovery rates and lower mortality ($p=0.003$), while visiting three or more healthcare facilities prior to definitive care was linked to increased disability ($p=0.003$).¹² This finding reinforces the clinical imperative for direct and rapid transport to facilities equipped with ASV and ICU capability, bypassing intermediate facilities that may further delay definitive treatment — a lesson directly applicable to rural Sumatera settings.

Pathophysiology of Neurotoxic Cobra Envenomation

Cobra venom from *Naja* species is a complex mixture of proteins with multiple toxic mechanisms. The primary neurotoxic fraction consists of long-chain and short-chain α -neurotoxins (α -Ntx), members of the three-finger toxin (3FTx) family. These α -neurotoxins selectively bind with high affinity to the α -subunit of muscle-type nicotinic acetylcholine receptors (nAChR) at the neuromuscular junction, producing a competitive, reversible post-synaptic blockade that prevents acetylcholine binding and subsequent muscle depolarisation.^{5,6}

Cryo-EM structural studies have elucidated the precise binding mode of short-chain α -neurotoxins, demonstrating that they are 'pinched' between loop C on the principal subunit and a hairpin in loop F on the complementary subunit, effectively occluding the neurotransmitter binding site.⁶ Unlike pre-synaptic neurotoxins (as in krait envenomation which cause axon terminal degeneration), the post-synaptic blockade by cobra α -neurotoxins is theoretically reversible with antivenom neutralisation, making timely antivenom administration of particular importance.

In addition to neurotoxins, *N. sumatrana* venom contains phospholipase A2 (PLA2), cardiotoxins, and cytotoxins. Cytotoxins are responsible for local tissue necrosis at the

envenomation site, while phospholipases contribute to membrane disruption and may exacerbate systemic inflammation.⁷ In the present case, the predominance of neurotoxic manifestations (progressive somnolence, declining GCS, respiratory failure) over local cytotoxic effects (localised oedema at bite site without early necrosis) suggests a primarily neurotoxin-dominant venom envenomation profile.

The neurotoxic clinical syndrome in cobra envenomation characteristically progresses in a descending pattern: ptosis → ophthalmoplegia → facial paresis → bulbar dysfunction → respiratory muscle paralysis.¹³ In the present case, the advanced stage of neurotoxic progression to respiratory failure at 8 hours post-envenomation is consistent with published kinetic data on *N. sumatrana* venom toxin absorption. Emesis (4 episodes) may reflect both autonomic dysregulation and direct toxic effects on the gastrointestinal nervous plexus.

Anti-Snake Venom (ASV): Role, Dosing, and Repeated Administration

Intravenous ASV remains the only specific, evidence-based treatment for neurotoxic cobra envenomation.¹ Snakebite envenomation treatment guidelines universally recommend prompt ASV administration as the first priority in managing systemic signs of envenomation, including neurotoxicity and respiratory compromise.

A randomised controlled trial conducted in Nepal comparing low-dose (2 vials) versus high-dose (10 vials) initial ASV for neurotoxic envenomation found no significant difference in the primary composite outcome (death, need for assisted ventilation, or worsening/recurrence of neurotoxicity) between the two dosing strategies (48.7% vs. 38.5%, $p=0.264$).¹⁴ However, the trial highlighted that the antivenom used in the region performed poorly overall, and that patients bitten by kraits fared worse than those bitten by cobras — a distinction relevant to dosing calculations for cobra envenomation.

In the present case, initial ASV availability was limited to 2 vials at the first-contact facility, representing a critical resource constraint in a secondary rural hospital. Upon ICU admission, the anesthesiologist protocol mandated 8 vials in 100 mL dextrose 5% over 2 hours, with serial 2-vial redosing at 6-hour intervals in the absence of clinical improvement, targeting a cumulative 24-hour total of 20 vials. This escalating repeated-dose strategy aligns with evidence-based

460

recommendations for monitoring and re-dosing ASV in patients who fail to demonstrate adequate clinical response to initial therapy.

Evidence from a prospective study in Cameroon demonstrated that sequential administration of low doses of antivenom, assessed at short intervals with renewal based on persistence of neurotoxicity, achieved complete cessation of neurotoxicity in 93% of patients within 24 hours while preserving antivenom supply.¹⁵ These findings support the titrated repeat-dosing strategy employed in this case.

Regarding adverse reactions to ASV, both acute anaphylactic and delayed serum sickness-type reactions are well-recognised complications of animal-derived ASV administration.¹⁶ Acute anaphylactic reactions typically manifest within 1 hour of ASV infusion. In the present case, no clinically documented adverse reactions to ASV were recorded during the admission, which may reflect the benefit of slow infusion and close monitoring in the ICU setting.

Acute Respiratory Failure: Diagnosis and Management

Acute respiratory failure secondary to neurotoxic cobra envenomation develops as a consequence of progressive respiratory muscle paralysis (primarily diaphragmatic and intercostal muscle involvement), compounded by bulbar dysfunction impairing airway protection reflexes.¹³ The patient in this case presented with tachypnoea (RR 23 bpm), declining GCS (E3M6V4), and chest pain — constituting an imminent respiratory crisis requiring urgent airway intervention.

The decision to proceed with orotracheal intubation was appropriate and timely. Early endotracheal intubation in patients with acute hypoxaemic respiratory failure has been associated with significantly reduced one-year mortality (HR 0.81; 95% CI 0.68–0.96) in large observational studies.¹⁷ Lung-protective mechanical ventilation strategies are recommended to minimise ventilator-induced lung injury and associated systemic inflammation.

The intensive care management in this case followed standard multidisciplinary ICU principles: mechanical ventilation with appropriate settings, haemodynamic monitoring, broad-spectrum antibiotic prophylaxis with meropenem (targeting gram-negative organisms including potential aspiration-related pathogens), analgesia with ketorolac, central venous access, and rigorous input-output monitoring.

Meropenem was administered at 3×1 g/day, which constitutes appropriate empiric broad-spectrum antibiotic coverage for potential aspiration pneumonia or secondary respiratory tract infection in a ventilated patient with a 8-hour pre-hospital period in a rural/agricultural environment. Gram-negative organisms, including *Morganella morganii*, *Proteus* spp., and *Pseudomonas aeruginosa*, are frequently implicated in wound infections following cobra bites, as demonstrated in studies of *Naja atra* bite bacteriology.¹⁸

Multidisciplinary ICU Outcomes

The patient demonstrated satisfactory clinical response to the combined therapeutic approach of mechanical ventilation support and high-dose repeated ASV. This is consistent with published cohort data from Malaysia on *Naja* species envenomation, wherein antivenom was administered in 30.7% of confirmed cases, with 78.3% receiving Thai Red Cross Cobra Antivenom and four deaths documented only due to secondary complications rather than primary neurotoxicity.⁴

Among King Cobra (*Ophiophagus hannah*) bites — the most severe elapid envenomation in Southeast Asia — tracheal intubation and ventilatory support were required in 40.6% of patients, with the majority achieving clinical recovery following antivenom administration.¹⁹ While *N. sumatrana* venom is generally less potent than *O. hannah*, the prolonged exposure due to 8-hour delay in the present case effectively amplified the envenomation severity to a comparably life-threatening level.

Clinical Lessons and Public Health Implications

This case generates several critical clinical lessons applicable to regional emergency practice:

1. Delayed hospital presentation is the primary modifiable risk factor in snake envenomation mortality and morbidity. Community education campaigns and standardised rapid referral protocols are essential.²⁰
2. Rural secondary hospitals in high-risk regions must maintain an adequate ASV stockpile. The initial availability of only 2 vials at the first-contact facility represents a dangerous resource constraint that directly impacts patient prognosis. WHO guidelines and national snakebite

management protocols should mandate minimum ASV reserves at district-level facilities in endemic zones.

3. Progressive GCS decline and respiratory rate elevation in the context of elapid envenomation must be recognised as signs of impending respiratory failure, mandating urgent anaesthesiology consultation and pre-emptive preparation for emergency intubation.

4. The neuromuscular blockade produced by cobra α -neurotoxins may be partially amenable to anticholinesterase agents (neostigmine/edrophonium), which have demonstrated benefit in improving bulbar function and respiratory capacity in monocled cobra envenomation.²¹ This pharmacological adjunct was not documented in the present case but should be considered in future cases where mechanical ventilation is not yet indicated but neurotoxic signs are progressive.

5. Healthcare knowledge gaps remain substantial: a systematic review and meta-analysis found that only 56% of healthcare workers and the general population answered knowledge questions about snakebite management correctly.²² Targeted educational interventions, as demonstrated in Tamil Nadu where community programs resulted in faster hospital arrival and better first aid, should be implemented in agricultural regions of Indonesia.

CONCLUSION AND RECOMMENDATIONS

This case report documents the clinical trajectory and successful management of severe neurotoxic envenomation following yellow cobra (*Naja sumatrana*) bite with an 8-hour pre-hospital delay, resulting in progressive systemic neurotoxicity culminating in acute respiratory failure requiring mechanical ventilation and high-dose cumulative anti-snake venom (ASV) therapy in the ICU. The case demonstrates that delayed presentation substantially amplifies envenomation severity, facilitating advanced toxin distribution and neuromuscular blockade that may otherwise be manageable with early antivenom administration.

The favourable clinical outcome in this case was attributable to: (1) timely recognition of impending respiratory failure and prompt orotracheal intubation; (2) escalating repeated-dose ASV protocol with appropriate monitoring; (3) broad-spectrum antibiotic coverage with meropenem; (4) multidisciplinary ICU management including mechanical ventilation, haemodynamic monitoring,

and central venous access; and (5) collaborative care between emergency medicine and anaesthesiology specialists.

Recommendations:

1. Regional health authorities should mandate minimum ASV stockpiles at all district-level hospitals in cobra-endemic zones throughout Sumatera.
2. Emergency physicians in rural settings should receive standardised training in the clinical recognition of progressive neurotoxic cobra envenomation and early intubation protocols.
3. Community-based snakebite education campaigns should emphasise the critical importance of immediate hospital transport, immobilisation of the bitten extremity, and avoidance of harmful first-aid practices (tourniquet, incision, suction).
4. Future prospective studies should document the pharmacokinetics of ASV neutralisation in delayed-presentation cobra envenomation to optimise repeated-dosing protocols in resource-limited ICU settings.
5. The development of region-specific, affordable polyvalent antivenom covering *N. sumatrana* and other medically significant Indonesian snake species should be prioritised by national and regional pharmaceutical authorities.

REFERENCES

1. Gutiérrez JM, Calvete JJ, Habib AG, Harrison RA, Williams DJ, Warrell DA. Snakebite envenoming. Nat Rev Dis Primers. 2017;3:17063. Available from: <https://consensus.app/papers/details/4b6531f6906d57bf8247d22ccbc6543f/>
2. Seifert SA, Boyer LV, Benson BE, Rogers JJ. Snake envenomation. N Engl J Med. 2022;387(7):e17. Available from: <https://consensus.app/papers/details/ee8ebb9f12e25fbf8de8105892cebe36/>
3. Zukifli NA, Azri AM, Ahmad SH, et al. In Vitro neurotoxicity and myotoxicity of Malaysian Naja sumatrana and Naja kaouthia venoms: Neutralization by monovalent and Neuro Polyvalent Antivenoms from Thailand. PLoS ONE. 2022;17(9):e0274521. Available from: <https://consensus.app/papers/details/6a84bd466b3c5b7da91657e7f7c6e9c6/>

4. Ismail AK, Ahmad AS, Hanapiah FM, et al. The frequency, clinical characteristics and outcomes of Naja species related injuries in Malaysia consulted to Remote Envenomation Consultancy Services from 2020–2023. *PLoS Negl Trop Dis.* 2025;19(1):e0012722. Available from: <https://consensus.app/papers/details/b48b640de6505d1bb0b76f81026e58e1/>
5. Ranawaka UK, Lalloo DG, de Silva HJ. Neurotoxicity in snakebite—the limits of our knowledge. *PLoS Negl Trop Dis.* 2013;7(10):e2302. Available from: <https://consensus.app/papers/details/6d6efc6682e055f5bb7ae46b1ddc2887/>
6. Nys M, Wijckmans E, Farinha A, et al. The molecular mechanism of snake short-chain α -neurotoxin binding to muscle-type nicotinic acetylcholine receptors. *Nat Commun.* 2022;13(1):4543. Available from: <https://consensus.app/papers/details/072194c7d23f5882a829ec6518d2a975/>
7. Yap MKK, Tan NH, Fung SY. Pharmacokinetics of Naja sumatrana (Equatorial Spitting Cobra) venom and its major toxins in experimentally envenomed rabbits. *PLoS Negl Trop Dis.* 2014;8(7):e2890. Available from: <https://consensus.app/papers/details/6914b3951cbd5ad3a1bfdfc43f88cba1/>
8. Mayeden S, Adu P, Afroz A, et al. Pediatric snakebite in Sub-Saharan Africa: Clinical predictors, outcomes, and gaps in care — A systematic review. *PLoS Negl Trop Dis.* 2026;20(1):e0012804. Available from: <https://consensus.app/papers/details/eeffe4ec6aa4598dae429c822d922db9/>
9. Afroz A, Engelman D, Lucas M, Steer A, Walls RS. Snakebite envenoming: a systematic review and meta-analysis of global morbidity and mortality. *PLoS Negl Trop Dis.* 2024;18(3):e0012043. Available from: <https://consensus.app/papers/details/22edf9b375f55126a282549667054dc0/>
10. Silva A, Pilapitiya S, Siribaddana S. Time delays in treatment of snakebite patients in rural Sri Lanka and the need for rapid diagnostic tests. *PLoS Negl Trop Dis.* 2020;14(4):e0008182. Available from: <https://consensus.app/papers/details/7727b9fe652d5b8db54973a29chec832a/>
11. Gerardo CJ, Quackenbush E, Lewis B, et al. Does this patient have a severe snake envenomation? The Rational Clinical Examination Systematic Review. *JAMA Surg.* 2019;154(4):373–381. Available from: <https://consensus.app/papers/details/9940465c2ef95fbc9942c7a5d12da930/>

12. Wagle U, Bhat V, Krishnamurthy S, et al. Pre-hospital interventions in snakebite: A telephonic survey and follow up investigating snakebite envenoming from a tertiary care centre in Coastal Karnataka. *PLoS Negl Trop Dis.* 2025;19(4):e0013003. Available from: <https://consensus.app/papers/details/a9bdce098f0b5adf9133774795554d32/>
13. Javed F, Malinowski M, Chen A, et al. 579: Acute descending paralysis: Decisions that come back to bite you. *Crit Care Med.* 2023;51(1):282. Available from: <https://consensus.app/papers/details/259e250ec3765029ad7adaff0c453b9d/>
14. Alirol E, Sharma SK, Bawaskar HS, Kuch U, Chappuis F. Dose of antivenom for the treatment of snakebite with neurotoxic envenoming: Evidence from a randomised controlled trial in Nepal. *PLoS Negl Trop Dis.* 2017;11(5):e0005612. Available from: <https://consensus.app/papers/details/2ed7b4844c4a543c81df9da55660a634/>
15. Chippaux JP, Massougboji A, Habib AG, et al. Real life condition evaluation of Inoserp PAN-AFRICA antivenom effectiveness in Cameroon. *PLoS Negl Trop Dis.* 2023;17(6):e0011415. Available from: <https://consensus.app/papers/details/843631aa910e587fa8096cacd02bbf30/>
16. Buckley NA, Isbister GK, Stiles MK, Jansen J. Adverse reactions to snake antivenom, and their prevention and treatment. *Br J Clin Pharmacol.* 2016;81(3):446–452. Available from: <https://consensus.app/papers/details/43760321eed95ed38801bd1cbfe735e2/>
17. Mellado-Artigas R, Rodriguez-Garcia R, Barbas CSV, Artigas A, Ferrando C. Effect of immediate initiation of invasive ventilation on mortality in acute hypoxemic respiratory failure: a target trial emulation. *Crit Care.* 2024;28(1):62. Available from: <https://consensus.app/papers/details/a9d822d47d1757d8bfca71c69caf355b/>
18. Mao YC, Liu PY, Hung DZ, et al. Bacteriology of *Naja atra* snakebite wound and its implications for antibiotic therapy. *Am J Trop Med Hyg.* 2016;94(5):1129–1135. Available from: <https://consensus.app/papers/details/427513ec036455a582b04eeea874acfb/>
19. Renault A, Ismail AK, Nordiana AB, et al. Frequency, geographical distribution, clinical characteristics, antivenom utilisation and outcomes of King Cobra (*Ophiophagus hannah*) bites in Malaysia. *PLoS Negl Trop Dis.* 2024;18(2):e0012044. Available from: <https://consensus.app/papers/details/bac8903543f6512c9338501718de4db5/>

20. Samuel S, Mathew P, Preetilal R, et al. Venomous snakebites: Rapid action saves lives — A multifaceted community education programme increases awareness about snakes and snakebites among the rural population of Tamil Nadu, India. *PLoS Negl Trop Dis*. 2020;14(11):e0008882. Available from: <https://consensus.app/papers/details/f9e05a327da15568b0f24715acd42fe5/>
21. Faiz MA, Ghose A, Ahsan MF, et al. Bites by the monocled cobra, *Naja kaouthia*, in Chittagong Division, Bangladesh: Epidemiology, clinical features of envenoming and management of 70 identified cases. *Am J Trop Med Hyg*. 2017;96(4):876–884. Available from: <https://consensus.app/papers/details/498965aa774758e19a3a4dace7925ff5/>
22. Afroz A, Engelman D, Lucas M, Steer A, Walls RS. Assessing knowledge and awareness regarding snakebite and management of snakebite envenoming in healthcare workers and the general population: A systematic review and meta-analysis. *PLoS Negl Trop Dis*. 2023;17(10):e0011679. Available from: <https://consensus.app/papers/details/516e06e8e6605e1cb0f31af8bf34ef02/>