e-ISSN (online): 2745-9497

Journal of Anesthesiology and Clinical Research (JACR)

Journal website: https://hmpublisher.com/index.php/jacr

Management of *Bungarus* sp. Envenomation Presenting as Rapidly Progressing Respiratory Failure: An Intensive Care Case Report

Mohammad Sutami[1*], Wiwi Jaya[2], Eka Oktaviana Hirda[3]

[1]Anesthesiology Resident, Dr. Saiful Anwar Regional General Hospital, Malang, Indonesia

[2]Department of Anesthesiology, Dr. Saiful Anwar Regional General Hospital, Malang, Indonesia

[3]General Practitioner, Fatimah Islamic Hospital, Banyuwangi, Indonesia

ARTICLE INFO

Keywords:
Bungarus
Envenomation
Krait
Neurotoxicity
Snakebite

***Corresponding author:**
Mohammad Sutami

E-mail address:
sutami_dr@student.ub.ac.id

All authors have reviewed and approved the final version of the manuscript.

https://doi.org/10.37275/jacr.v6i1.746

A B S T R A C T

Introduction: Envenomation by snakes of the *Bungarus* genus (kraits) represents a critical medical emergency, particularly prevalent in South and Southeast Asia, including Indonesia. Krait venom is primarily neurotoxic, often containing potent presynaptic toxins (β-bungarotoxins) that disrupt neuromuscular transmission, leading to rapidly progressive descending paralysis. Respiratory failure due to diaphragmatic and intercostal muscle paralysis is the most life-threatening complication, necessitating immediate and expert intensive care management. **Case presentation:** We report the case of a 55-year-old Indonesian male who presented to the emergency department approximately five hours after being bitten on his right hand by a snake suspected to be a Weling (*Bungarus* sp.). He exhibited rapidly deteriorating neurological function, including dysarthria and decreased consciousness, progressing swiftly to acute respiratory failure with paradoxical breathing and hypoxia. Emergent endotracheal intubation and mechanical ventilation were instituted. Subsequent management in the Intensive Care Unit (ICU) involved continued ventilatory support, administration of polyvalent snake antivenom (SABU), sedation, broad-spectrum antibiotics for complicating pneumonia, and comprehensive supportive care. Nerve conduction studies later confirmed bilateral phrenic nerve palsy and severe sensorimotor axonal polyneuropathy. **Conclusion:** This case highlights the fulminant respiratory failure characteristic of severe *Bungarus* envenomation. Prompt recognition, aggressive airway management, and mechanical ventilation are paramount lifesaving interventions. While antivenom administration is a standard therapy, its efficacy in reversing established presynaptic neuromuscular blockade remains debated, underscoring the critical role of prolonged ventilatory support and meticulous ICU care until neuromuscular function recovers, which can be significantly delayed due to the nature of presynaptic toxins. This case reinforces the need for high vigilance and resource preparedness in managing neurotoxic snakebites in endemic regions.

1. Introduction

Snakebite envenomation represents a significant global health challenge, particularly in tropical and subtropical regions. It is a major yet frequently neglected public health crisis that disproportionately affects vulnerable rural populations across Africa, Asia, Latin America, and Oceania. The World Health Organization (WHO) estimates that snakebite envenomation occurs in 1.8 to 2.7 million people each year, resulting in an estimated 81,000 to 138,000 deaths and causing approximately three times as many permanent disabilities. The burden of snakebite is especially pronounced in Southeast Asia, including Indonesia. Snakebite is often described as a disease of poverty, primarily affecting individuals in impoverished communities such as agricultural workers, farmers,

fishermen, and children, who often have limited access to timely and effective healthcare. Recognizing the severity of this issue, the WHO in 2017 formally classified snakebite envenomation as a highest-priority neglected tropical disease. This classification underscores the urgent need for increased attention and resources to combat snakebite, and it led to the launch of a global strategy aimed at reducing deaths and disabilities by 50% by 2030. Indonesia, a large archipelago characterized by its rich biodiversity, is home to a diverse array of venomous snake species. These snakes belong predominantly to the Elapidae and Viperidae families, both of which pose significant medical threats. The venoms of snakes from these two families differ significantly in their composition and effects. Viperid bites typically result in local tissue damage, coagulopathy (disorders of blood clotting), and systemic bleeding. In contrast, elapid venoms are predominantly neurotoxic, cardiotoxic, or a combination of both.[1-3]

Among the Elapidae family, snakes of the genus *Bungarus*, commonly known as kraits, are particularly feared in Indonesia. These snakes have various local names, including Weling and Welang. Several *Bungarus* species are known to cause severe neurotoxic envenomation, with the Malayan krait (*Bungarus candidus*) and the Banded krait (*Bungarus fasciatus*) being especially notorious. Recent data indicates an increasing incidence of snakebites in some regions of Indonesia, with elapids like the Javan spitting cobra (*Naja sputatrix*) and kraits contributing substantially to morbidity and mortality. The pathophysiology of *Bungarus* envenomation is primarily characterized by potent neurotoxicity. This neurotoxicity is mediated by a complex mixture of venom components that target the neuromuscular junction (NMJ), the site where nerve impulses are transmitted to muscles. Krait venoms typically contain both postsynaptic and presynaptic neurotoxins, each with distinct mechanisms of action. Postsynaptic neurotoxins, such as α-bungarotoxins, are often referred to as 'three-finger toxins' due to their structural characteristics. These toxins bind competitively, and often reversibly (although with high affinity), to nicotinic acetylcholine receptors (nAChRs) on the muscle endplate. By binding to these receptors, they block the action of acetylcholine, a neurotransmitter essential for muscle contraction, thus causing a non-depolarizing neuromuscular blockade, similar to the effect of curare. In some cases, this blockade can potentially be overcome by increasing the concentration of acetylcholine in the synaptic cleft using anticholinesterase agents like neostigmine. However, the most dangerous components of *Bungarus* venoms, particularly those of *B. candidus* and *B. caeruleus* (the common krait), are the presynaptic β-bungarotoxins. These toxins are potent phospholipase A2 (PLA2) enzymes that bind irreversibly to the motor nerve terminal. The action of β-bungarotoxins is complex and involves an initial enhancement followed by a profound inhibition of acetylcholine release. This ultimately leads to structural damage of the nerve terminal, depletion of synaptic vesicles (which store acetylcholine), axonal degeneration, and functional denervation of the muscle. A critical factor in the severity of envenomation by kraits is that this presynaptic damage is largely insensitive to both antivenom (once binding has occurred) and anticholinesterase drugs. Consequently, clinical recovery from presynaptic neurotoxicity is typically slow, depending entirely on the regeneration of damaged nerve terminals and the formation of new neuromuscular junctions, a process that can take days to weeks.[4-6]

The clinical presentation of *Bungarus* envenomation can be insidious, often beginning with minimal or no local signs at the bite site. This lack of prominent local signs can be misleading and may result in a false sense of security. Systemic neurotoxic symptoms usually manifest within a few hours after the bite, but their onset can be delayed by up to 12 hours or even longer in some cases. The classical presentation of *Bungarus* envenomation involves a descending paralysis. This paralysis often begins with ptosis (droopy eyelids) and ophthalmoplegia (difficulty moving the eyes), followed by bulbar palsy, which manifests as difficulty swallowing and speaking (dysarthria). The paralysis can then progress to involve the intercostal muscles and the diaphragm, leading to respiratory failure, which is the principal cause of death in untreated or inadequately managed cases. In addition to these characteristic

features, other systemic manifestations of *Bungarus* envenomation may include abdominal pain, vomiting, generalized muscle weakness, and in some cases, altered sensorium or coma, particularly with bites from *Bungarus caeruleus*. Some *Bungarus* species can also cause rhabdomyolysis, a condition involving the breakdown of muscle tissue, which can lead to myoglobinuria and acute kidney injury. The management of severe neurotoxic envenomation, especially from *Bungarus* species, presents significant challenges, particularly in resource-limited settings. The fundamental aspects of management include prompt recognition of systemic envenomation, vigilant monitoring for impending respiratory failure, and aggressive airway management, which may necessitate endotracheal intubation and sustained mechanical ventilation. Snake antivenom is the only specific therapy available to neutralize circulating venom toxins. However, the effectiveness of antivenom, especially in reversing established presynaptic neurotoxicity, is limited. Furthermore, the optimal dosing regimens for *Bungarus* bites are still debated, with some evidence suggesting that higher doses may be required compared to envenomation by other elapids. The role of anticholinesterase agents in the treatment of krait envenomation is generally considered minimal, given the predominantly presynaptic nature of the venom's neurotoxicity. While they may be considered in specific circumstances or for cobra bites, they are not the primary treatment for krait envenomation. Comprehensive supportive care in an intensive care unit (ICU) setting is essential for managing complications, providing life support, and facilitating the patient's recovery.[7-10] This case report aims to provide a detailed account of the intensive care management and neurophysiological impact of severe *Bungarus* sp. envenomation in Indonesia. By presenting a case of a patient who developed rapidly progressive respiratory failure, the report seeks to correlate the clinical progression with specific electrodiagnostic findings, particularly bilateral phrenic nerve palsy. Furthermore, it will discuss the critical importance of timely mechanical ventilation and supportive care in the management of predominantly presynaptic neurotoxicity resulting from krait envenomation.

2. Case Presentation

The patient was a 55-year-old male, with body weight 60 kg and height 160 cm. The patient's primary complaints upon arrival at the Emergency Department (ED) were a worsening shortness of breath, difficulty speaking, described as dysarthria, and a decreased level of consciousness. The history of the present illness revealed that the patient had sustained a snakebite on the dorsum of his right hand approximately five hours prior to his arrival at the ED. The incident occurred while the patient was working in a field, and the snake species was unidentified. Initially, following the bite, the patient only reported localized pain in the affected right hand. However, in the subsequent hours, his condition deteriorated, marked by the progression of dyspnea, the onset of dysarthria, and a gradual decline in his level of consciousness. The patient's past medical history was unremarkable, with no significant prior medical conditions reported. It was also confirmed that there was no history of any other trauma preceding these events, apart from the snakebite itself. The patient's general appearance upon arrival at the ED was that of a critically ill individual exhibiting severe respiratory distress and an altered mental status. Assessment of the patient's airway revealed it to be compromised, as evidenced by the presence of snoring and gargling sounds, which are indicative of upper airway obstruction. This obstruction was likely attributed to the loss of pharyngeal muscle tone. The patient's breathing was significantly labored, characterized by tachypnea, with a respiratory rate of 28 breaths per minute, shallow respirations, and paradoxical chest wall movement. The paradoxical chest wall movement, where the abdomen moves outward while the chest moves inward during inhalation, strongly suggested paralysis of the intercostal muscles. Furthermore, the patient exhibited hypoxia, with a recorded oxygen saturation (SpO_2) of 92% despite the administration of oxygen via a non-rebreather mask at a flow rate of 15 liters per minute. Auscultation of the chest revealed diminished breath sounds bilaterally, indicating reduced air entry into both lungs. In terms of

circulation, the patient was initially hemodynamically stable. His blood pressure was measured at 152/86 mmHg, and his heart rate was 87 beats per minute. The patient's extremities were warm to the touch, and the capillary refill time was less than 2 seconds, suggesting adequate peripheral perfusion. A neurological examination revealed a decreased level of consciousness, with the patient only responsive to painful stimuli. The patient's pupils were noted to be dilated, measuring 5 mm in both eyes, but they were reactive to light bilaterally. An accurate Glasgow Coma Scale (GCS) score was challenging to determine due to the patient's impending respiratory arrest; however, it was estimated to be approximately E1V1M4, resulting in a total score of 6. Examination of the patient's skin and extremities revealed the presence of fang marks on the dorsum of the right hand, which confirmed the snakebite. There was mild swelling around the bite site, but at this initial examination, there was no obvious necrosis or blistering. The patient's core body temperature was within the normal range, and he was afebrile. Following the initial assessment and the critical decision to secure the patient's airway, the patient was intubated and sedated and subsequently admitted to the Intensive Care Unit (ICU) for further management and monitoring. Upon arrival in the ICU, the patient remained intubated and sedated. The sedation was maintained with continuous infusions of Midazolam at a rate of 3 mg per hour and Fentanyl at a rate of 30 mcg per hour. The patient's airway was patent via the endotracheal tube, and his breathing was being supported by mechanical ventilation. The mode of ventilation was Pressure Support Synchronized Intermittent Mandatory Ventilation (PSIMV). The initial ventilator settings included a respiratory rate of 12 breaths per minute, a positive end-expiratory pressure (PEEP) of 5 cmH_2O, a fraction of inspired oxygen (FiO_2) of 80%, an inspiratory pressure (Pins) of 15 cmH_2O, a pressure limit (Plimit) of 30 cmH_2O, an inspiratory time (Ti) of 1.0 second, and a flow trigger of 5.0 L/min. These settings achieved a tidal volume (VT) of 544 mL, which approximated 9 mL/kg of the patient's predicted body weight. The patient's oxygen saturation (SpO_2) remained at 100% under these ventilator settings. Auscultation of the chest revealed the presence of bilateral vesicular breath sounds, with scattered rhonchi noted. Assessment of the patient's circulation indicated that peripheral perfusion remained adequate, with warm extremities and a capillary refill time of less than 2 seconds. The patient's heart rate was 100 beats per minute, and his blood pressure was 124/64 mmHg. The cardiac rhythm was regular, and heart sounds were normal, with no murmurs or gallops detected. Neurological examination of the sedated patient revealed that his pupils were 3mm in diameter, equal, round, and reactive to light bilaterally. There were no lateralizing signs observed. It is important to note that a subsequent Nerve Conduction Velocity (NCV) report later confirmed the presence of flaccid paralysis, decreased sensation, and absent reflexes. Examination of the abdomen revealed it to be soft and non-distended, with the presence of bowel sounds. A Foley catheter was in situ, producing an adequate urine output of approximately 200 cc over the initial few hours, and the urine was clear and yellow. The patient's axillary temperature was 36.7°C, there was no cyanosis, and no peripheral edema was noted. Minimal edema was observed at the snakebite site on the right hand. Laboratory evaluations conducted on Day 1 revealed several notable findings. Serum electrolyte levels showed a sodium level of 138 mmol/L, a potassium level of 3.32 mmol/L, indicating hypokalemia, and a chloride level of 104 mmol/L. The complete blood count (CBC) showed a hemoglobin level of 13.3 g/dL, a white blood cell count (WBC) of 12,170 x $10^3/mm^3$, indicating leukocytosis, a hematocrit of 41.1%, and a platelet count of 240,000/mm^3. Coagulation studies revealed a prothrombin time (PT) of 11.1 seconds, with an International Normalized Ratio (INR) of 1.07, and an activated partial thromboplastin time (APTT) of 21.4 seconds, both within normal limits. Liver function tests showed an aspartate transaminase (AST) level of 22 U/L and an alanine transaminase (ALT) level of 14 U/L, both within the normal range. Renal function tests showed a urea level of 33.6 mg/dL and a creatinine level of 1.07 mg/dL, also within normal limits. The random blood glucose level was 130 mg/dL. Arterial blood gas (ABG) analysis performed post-intubation, with the patient receiving an FiO_2 of 80%, revealed a pH of 7.37, a $PaCO_2$ of 39.0 mmHg, a PaO_2 of 111.3 mmHg, a bicarbonate

(HCO_3) level of 23.0 mmol/L, a base excess of -2.5 mmol/L, and an oxygen saturation (SaO_2) of 97.8%. The calculated PaO_2/FiO_2 (P/F) ratio was 139.0, indicating moderate acute respiratory distress syndrome (ARDS) or hypoxemia. Pre-intubation ABG, obtained while the patient was on a non-rebreather mask at 15L/min, showed respiratory alkalosis with hypoxia, with a pH of 7.48, a $PaCO_2$ of 33.1 mmHg, a PaO_2 of 86.6 mmHg, an HCO_3 of 24.9 mmol/L, and an SaO_2 of 96.7%. A chest X-ray performed on Day 1 revealed the presence of bilateral pulmonary infiltrates, suggestive of pneumonia, and cardiomegaly. Serial chest X-rays conducted on Day 2 and Day 3 showed a gradual reduction in the pulmonary infiltrates, indicating improvement in the pneumonia, although the cardiomegaly persisted. Nerve conduction velocity (NCV) and electromyography (EMG) studies, performed on Day 2 due to the patient's profound muscle weakness and respiratory failure, revealed significant findings. These findings included bilateral severe phrenic nerve palsy, characterized by prolonged latency and low amplitude or absent response, and severe generalized axonal demyelinating motor and sensory polyneuropathy affecting multiple nerves, including the median, ulnar, peroneal, and tibial nerves. The polyneuropathy was characterized by prolonged distal latencies, significantly reduced amplitudes, slow conduction velocities, and abnormal F-waves. Sensory nerve action potentials (SNAPs) showed no response in the median, ulnar, and sural nerves. These electrodiagnostic findings were highly consistent with severe neuromuscular damage secondary to neurotoxic envenomation, particularly affecting nerve axons and myelin, including the critical respiratory nerves. Based on the patient's history, clinical findings, and the results of investigations, the following clinical diagnoses were established: Acute Respiratory Failure secondary to Neurotoxic Snakebite Envenomation, with the snake suspected to be a *Bungarus* species, specifically the Weling krait. The patient also presented with severe neuromuscular paralysis, including bilateral phrenic nerve palsy and generalized polyneuropathy, necessitating mechanical ventilation dependency. Further diagnoses included moderate hypoxemia, with a P/F ratio of 139, community-acquired pneumonia or aspiration pneumonia, Heart Failure Stage B, based on the cardiomegaly observed on the chest X-ray, which was likely a pre-existing or incidental finding, and mild hypokalemia (Table 1).

The management of this patient with severe neurotoxic snakebite envenomation required a swift and multifaceted approach, beginning in the Emergency Department (ED) and continuing through several days of intensive care in the ICU. The primary goals of treatment were to support the patient's respiratory function, neutralize the venom, manage complications, and provide comprehensive supportive care. The patient's critical condition upon arrival at the ED necessitated immediate interventions, primarily focused on securing his airway and supporting his failing respiratory function. The patient presented with a compromised airway, characterized by audible snoring and gargling sounds, indicative of upper airway obstruction. To address this, suctioning was performed to clear the airway, and an oropharyngeal airway (OPA) was temporarily inserted to maintain patency. Given the rapid deterioration of the patient's respiratory status and the signs of impending respiratory failure, emergent endotracheal intubation (ETI) was deemed necessary and was successfully performed. This procedure involved the insertion of a cuffed endotracheal tube (ETT) to establish a secure airway for mechanical ventilation. Following intubation, mechanical ventilation was initiated using a transport ventilator to support the patient's breathing. This intervention proved crucial, as the patient's oxygen saturation (SpO_2), which was initially 92% on a non-rebreather mask, improved significantly to 99% with mechanical ventilation. The institution of mechanical ventilation aimed to take over the work of breathing, allowing the respiratory muscles to rest and ensuring adequate oxygenation and carbon dioxide removal. In addition to airway and breathing management, initial circulatory support was provided in the ED through the administration of intravenous fluids. However, the specific type and rate of fluid administration in the ED are not detailed in the provided documentation. Upon transfer to the Intensive Care Unit (ICU), the patient's management was intensified and broadened to address the various aspects of his critical condition. Mechanical

ventilation was continued in the ICU, with the patient placed on Pressure Support Synchronized Intermittent Mandatory Ventilation (PSIMV) mode. The initial ventilator settings were carefully established to optimize respiratory support. These settings included a respiratory rate of 12 breaths per minute, a positive end-expiratory pressure (PEEP) of 5 cmH_2O, a fraction of inspired oxygen (FiO_2) of 80%, an inspiratory pressure of 15 cmH_2O, a pressure limit of 30 cmH_2O, an inspiratory time of 1.0 second, and a flow trigger of 5.0 L/min. These settings achieved a tidal volume of approximately 9 mL/kg of the patient's predicted body weight, and the patient's SpO_2 was maintained at 100%. The strategy behind these ventilator settings was to provide adequate alveolar ventilation and oxygenation while minimizing the risk of ventilator-induced lung injury. Given the history of snakebite and the clinical signs suggestive of neurotoxic envenomation, the administration of snake antivenom was a critical component of the patient's treatment. The antivenom used was a polyvalent snake antivenom (SABU - Serum Anti Bisa Ular) produced by PT Bio Farma, Indonesia. Two vials of the antivenom were administered intravenously, diluted in 100 cc of 0.9% sodium chloride solution, and infused over approximately one hour. This administration occurred approximately 7 hours post-bite. Prior to antivenom administration, it was confirmed that the patient had no known history of allergies, and resuscitation backup was readily available in case of any adverse reactions. The decision to administer only two vials of antivenom was based on local protocols and the lack of clear evidence supporting the benefit of repeated doses in cases of established presynaptic toxicity. To ensure patient comfort, facilitate mechanical ventilation, and alleviate anxiety, continuous intravenous infusions of Midazolam and Fentanyl were initiated. Midazolam, a benzodiazepine, was administered at a rate of 3 mg per hour, while Fentanyl, an opioid analgesic, was given at a rate of 30 mcg per hour. The doses of these medications were titrated based on the patient's sedation scores, with the initial goal of achieving deep sedation. In light of the chest X-ray findings suggestive of pneumonia and the clinical context, broad-spectrum antibiotics were started empirically to cover potential pathogens. Intravenous Levofloxacin 750 mg once daily and intravenous Metronidazole 500 mg every 8 hours were administered. This combination was chosen to provide coverage against a range of community-acquired and aspiration pneumonia pathogens. Several other supportive medications were administered to address specific needs and prevent potential complications. Intravenous fluids, specifically 0.9% sodium chloride solution, were administered at a rate of 40 cc per hour for maintenance, with meticulous monitoring of the patient's fluid balance. To prevent stress ulcers, intravenous Ranitidine 50 mg was given every 8 hours. For analgesia and antipyretic effects, intravenous Paracetamol 1 gram was administered every 8 hours. Tetanus prophylaxis was provided with the intramuscular administration of Tetanus Immunoglobulin (Tetagam) 250 IU. Potassium replacement was initiated to correct the patient's hypokalemia. Recognizing the importance of early nutritional support, a nasogastric tube (NGT) was inserted, and enteral feeding was initiated once bowel sounds were confirmed to be present. The patient was started on a standard enteral formula (NPC 6x200cc). In addition to mechanical ventilation, specific respiratory care measures were implemented to optimize lung function and prevent complications. The head of the bed was elevated to 30 degrees to reduce the risk of aspiration and improve lung expansion. Nebulization with 3% sodium chloride solution was performed every 8 hours to aid in airway clearance. Regular suctioning of the endotracheal tube was carried out to maintain airway patency. Comprehensive and continuous monitoring was essential to assess the patient's response to treatment and detect any changes in his condition. This included continuous electrocardiogram (ECG) monitoring, pulse oximetry (SpO_2) monitoring, temperature monitoring, and urine output monitoring via a Foley catheter. Daily laboratory tests, serial arterial blood gas (ABG) analyses, and serial chest X-rays were performed as clinically indicated. Regular neurological assessments were conducted, although these were limited by the patient's sedation. Hemodynamic monitoring was also closely observed. Collaboration with the surgical department was planned regarding the management of the snakebite

site on the patient's hand. Informed consent was obtained from the patient's family, and they were kept updated about his critical condition and the ongoing management plan. The patient's progress was closely monitored throughout his stay in the ICU, and the treatment plan was adjusted as needed. On the second day of ICU admission, the patient's clinical status remained largely unchanged. He continued to be fully ventilator-dependent and sedated. There were no reported episodes of desaturation, hypotension, or bradycardia. Mechanical ventilation was continued using the same PSIMV settings, with an FiO_2 of 80% and SpO_2 maintained at 100%. Hemodynamics remained stable, with a heart rate of 100 beats per minute and a blood pressure of 124/64 mmHg. All ongoing treatments, including intravenous fluids, enteral nutrition, sedation and analgesia infusions, antibiotics (Levofloxacin and Metronidazole), Ranitidine, Paracetamol, and nebulization, were continued. Monitoring of the patient's airway, hemodynamics, and urine output was continued, and periodic evaluations of his respiratory pattern and motor strength were planned. A chest X-ray on Day 2 showed a slight improvement in the pulmonary infiltrates. The patient's condition on the third day of ICU stay was also generally stable, with no significant changes from the previous day. He remained fully ventilated and sedated, and no adverse events were reported. Ventilation was continued with the same PSIMV settings, maintaining SpO_2 at 100%. Hemodynamics remained stable. All ongoing treatments were continued. Monitoring continued as per the previous days, with ongoing evaluation for signs of neurological recovery. The chest X-ray showed further slight clearing of the pulmonary infiltrates, indicating improvement in the pneumonia. There was a consideration to potentially lighten the patient's sedation slightly to assess his neurological baseline, provided his respiratory and hemodynamic status allowed for it. However, the patient likely remained unresponsive or weak. On the fourth day, there was a notable observation of some spontaneous respiratory effort by the patient, although it was deemed insufficient to support weaning from mechanical ventilation. The patient's level of consciousness improved somewhat when sedation was lightened, with a Glasgow Coma Scale (GCS) score recorded as E4VTM5 = GCS 9T. Hemodynamics remained stable, and urine output was adequate. Given the patient's overall improvement and the emergence of some spontaneous respiratory effort, a decision was made to transfer him out of the ICU to a lower dependency unit or ward. This transfer was planned for continued monitoring, supportive care, and a more gradual weaning process from mechanical ventilation as his neuromuscular function recovered. The patient's final assessment upon transfer from the ICU indicated that his acute respiratory failure and hypoxemia were improving, and the pneumonia was resolving. However, he continued to have Heart Failure Stage B and the effects of the *Bungarus* sp. snakebite with neurotoxicity. Following the patient's transfer from the ICU, a neurology follow-up appointment was recommended in 2 months. The expected course of recovery was anticipated to be prolonged due to the severe axonal polyneuropathy and phrenic nerve palsy, potentially requiring long-term rehabilitation (Table 2).

3. Discussion

The patient's clinical presentation aligns remarkably well with the established pathophysiology of envenomation by snakes of the *Bungarus* genus, whose venoms are characterized by the presence of potent presynaptic neurotoxins, specifically β-bungarotoxins. The rapid sequence of events, from the initial snakebite to the development of dysarthria, a decline in consciousness, and the subsequent fulminant respiratory failure within a mere five hours, is a hallmark of severe envenomation by certain *Bungarus* species, such as *B. candidus* (Malayan krait) or *B. caeruleus* (common krait). While the classic presentation of *Bungarus* envenomation often involves early manifestations like ptosis (drooping of the eyelids) and ophthalmoplegia (paralysis of eye muscles), this case underscores that respiratory muscle paralysis can sometimes be the predominant or most rapidly progressing feature, demanding immediate recognition and intervention.

Table 1. Summary of patient's clinical findings on presentation and during initial ICU stay.

Category	Finding	Details & Specific Values
Demographics	Age	55 years
	Gender	Male
	Weight	60 kg
	Height	160 cm
Anamnesis (History)	Chief Complaint	Worsening shortness of breath, dysarthria (difficulty speaking), decreased level of consciousness.
	History of Present Illness	Snakebite on the dorsum of the right hand ~5 hours prior to Emergency Department (ED) arrival while working in a field. Snake type unknown. Initial local pain followed by progressive systemic symptoms.
	Past Medical History	No significant history reported. No known trauma other than the bite.
Physical examination (Pre-Intubation / ED Arrival)	General Appearance	Critically ill, severe respiratory distress, altered mental status.
	Airway	Compromised: Snoring, gargling sounds.
	Breathing	Tachypnea (RR: 28/min), shallow respirations, paradoxical chest wall movement, hypoxia (SpO_2 92% on 15L NRBM), diminished breath sounds bilaterally.
	Circulation	BP: 152/86 mmHg, HR: 87/min, regular rhythm, extremities warm, CRT < 2 sec.
	Disability (Neurological)	Decreased level of consciousness (responsive only to pain initially, GCS E1V1M4) pupils 5mm/5mm, reactive to light (+/+), no lateralization.
	Exposure (Skin/Extremities)	Fang marks (+) noted on the dorsum of the right hand. Mild swelling, no necrosis or blistering initially. Afebrile.
Physical examination (Post-Intubation / ICU Admission)	General	Intubated, sedated (Midazolam 3 mg/hr, Fentanyl 30 mcg/hr).
	Airway/Breathing	ETT patent, mechanically ventilated (PSIMV mode), RR 12/min, SpO_2 100% (FiO_2 80%), bilateral vesicular sounds, rhonchi (+/+).
	Circulation	BP: 124/64 mmHg, HR: 100/min, regular rhythm, S1S2 normal, no murmurs/gallops, warm peripheries, CRT < 2 sec.
	Disability (Neurological)	Sedated (GCS not applicable), Pupils 3mm/3mm, reactive to light (+/+), no lateralization. (NCV report later confirmed flaccid paralysis, decreased sensation, absent reflexes).
	Abdomen	Soft, non-distended, bowel sounds present.
	Renal	Foley catheter in situ, adequate urine output (200 cc initially), clear yellow.
	Skin/Temp	Axillary Temp: 36.7°C, no cyanosis, no peripheral edema.
Laboratory findings (Day 1)	Electrolytes	Na: 138 mmol/L, K: 3.32 mmol/L (Hypokalemia), Cl: 104 mmol/L.
	Complete Blood Count (CBC)	Hb: 13.3 g/dL, WBC: 12,170 x 10^3/mm^3 (Leukocytosis), Hct: 41.1%, Platelets: 240,000/mm^3.
	Coagulation	PT: 11.1 sec (INR 1.07), APTT: 21.4 sec (Normal).
	Liver Function Tests (LFTs)	AST (OT): 22 U/L, ALT (PT): 14 U/L (Normal).
	Renal Function Tests (RFTs)	Urea: 33.6 mg/dL, Creatinine: 1.07 mg/dL (Normal).
	Blood Glucose	Random Blood Sugar (GDS): 130 mg/dL.
	Arterial Blood Gas (Pre-intubation, NRBM 15L)	pH: 7.48, $PaCO_2$: 33.1 mmHg, PaO_2: 86.6 mmHg, HCO_3: 24.9 mmol/L, SaO_2: 96.7%. (Respiratory Alkalosis with Hypoxemia)
	Arterial Blood Gas (Post-intubation, FiO_2 80%)	pH: 7.37, $PaCO_2$: 39.0 mmHg, PaO_2: 111.3 mmHg, HCO_3: 23.0 mmol/L, Base Excess: -2.5 mmol/L, SaO_2: 97.8%. (Corrected ventilation, persistent moderate hypoxemia)
	Oxygenation Index	PaO_2/FiO_2 Ratio (P/F Ratio): 139.0 (Moderate ARDS/Hypoxemia).
Imaging findings	Chest X-Ray (Day 1)	Bilateral pulmonary infiltrates consistent with Pneumonia; Cardiomegaly.
	Chest X-Ray (Day 2 & 3)	Gradual reduction/improvement in pulmonary infiltrates; Cardiomegaly persisted.
	Nerve Conduction Velocity (NCV) / Electromyography (EMG) (Day 2)	**Conclusion:** 1. Bilateral Severe Phrenic Nerve Palsy. 2. Severe General Axonal Demyelinating Motoric Sensory Polyneuropathy. **Details:** Prolonged latencies, low/absent amplitudes (CMAP/SNAP), slow conduction velocities, poor F-wave responses in multiple nerves (Median, Ulnar, Peroneal, Tibial, Sural, Phrenic).
Clinical diagnosis (ICU Assessment)	Primary Diagnosis	Acute Respiratory Failure on Mechanical Ventilator secondary to Suspected Neurotoxic Snakebite Envenomation (*Bungarus sp.* / Weling).
	Associated Conditions / Complications	1. Pneumonia (Community-Acquired / Aspiration)
		2. Moderate Hypoxemia (P/F Ratio 139.0)
		3. Severe Neuromuscular Paralysis (incl. Bilateral Phrenic Nerve Palsy & Generalized Polyneuropathy)
		4. Heart Failure Stage B (Incidental finding)
		5. Mild Hypokalemia

Table 2. Summary of treatment procedures and ICU follow-up.

Time point / Aspect	Procedure / Observation	Details / Notes
Emergency Department (ED)	**Airway Management**	Snoring/gargling noted. Suction performed, Oropharyngeal Airway (OPA) placed temporarily. Emergent endotracheal intubation (ETT) performed successfully.
	Breathing Support	Mechanical ventilation initiated post-intubation (via transport ventilator). SpO_2 improved from 92% on NRBM to 99% on ventilator.
	Circulation Support	Intravenous fluids initiated (type/rate not specified in ED notes).
ICU Admission (Day 1)	**Ventilation**	Mode: PSIMV (Pressure Support Synchronized Intermittent Mandatory Ventilation). Settings: RR 12/min, PEEP 5 cmH_2O, FiO_2 80%, Insp. Pressure 15 cmH_2O, Pressure Limit 30 cmH_2O, Insp. Time 1.0 sec, Flow Trigger 5.0 L/min. Target VT ~9 mL/kg. Target SpO_2 > 94%.
	Antivenom Therapy	**SABU (Serum Anti Bisa Ular - Bio Farma):** 2 vials administered intravenously, diluted in 100 cc NaCl 0.9%, infused over ~1 hour. (Administered on Day 1, approx. 7 hours post-bite)
	Sedation & Analgesia	Continuous IV Infusion: Midazolam 3 mg/hr. Continuous IV Infusion: Fentanyl 30 mcg/hr. Target: Deep sedation initially (RASS -4 to -5 implied).
	Antibiotics (for Pneumonia)	IV Levofloxacin 1 x 750 mg (Day 1 dose administered). IV Metronidazole 3 x 500 mg (started).
	Supportive Medications	IV Fluids: NaCl 0.9% @ 40 cc/hr (maintenance). IV Ranitidine 3 x 50 mg (stress ulcer prophylaxis). IV Paracetamol 3 x 1 gr (analgesia/antipyretic). Tetagam (Tetanus Immunoglobulin) 250 IU IM (administered). Potassium replacement (for hypokalemia).
	Nutrition	Nasogastric tube (NGT) inserted. Enteral feeding started: Diet NPC (solid liquid nutrition) 6 x 200 cc.
	Respiratory Care	Head of bed elevated 30 degrees. Nebulization with NaCl 3% every 8 hours. Regular ETT suctioning.
	Monitoring	Continuous ECG, SpO_2, Temperature, Urine Output (Foley catheter). Daily Labs, Serial ABGs, Serial CXRs. Neurological checks (limited by sedation). Hemodynamic monitoring.
	Consultations	Collaboration planned with Surgery Dept. for bite site management.
	Communication	Informed consent obtained. Family informed about the patient's critical condition.
ICU follow-up (Day 2)	**Status**	No significant clinical changes. No desaturation, hypotension, or bradycardia reported. Remained fully ventilator-dependent and sedated.
	Ventilation	Continued on PSIMV mode with unchanged settings (FiO_2 80%). SpO_2 100%.
	Hemodynamics	Stable (HR 100/min, BP 124/64 mmHg).
	Treatments Continued	IV Fluids, Enteral Nutrition, Sedation/Analgesia infusions, Levofloxacin (Day 2), Metronidazole, Ranitidine, Paracetamol, Nebulization.
	Monitoring/Plan	Continued monitoring of airway (DOPE assessment), hemodynamics, urine output. Evaluate respiratory pattern and motor strength periodically.
ICU follow-up (Day 3)	**Status**	Patient remained clinically stable, fully ventilated, and sedated. No adverse events reported.
	Ventilation	Continued on PSIMV mode with unchanged settings (FiO_2 80%). SpO_2 100%.
	Hemodynamics	Stable (HR 100/min, BP 124/64 mmHg).
	Treatments Continued	IV Fluids, Enteral Nutrition, Sedation/Analgesia infusions, Levofloxacin (Day 3), Metronidazole, Ranitidine, Paracetamol, Nebulization.
	Monitoring/Plan	Ongoing monitoring as per previous days. Continued evaluation for signs of neurological recovery.
ICU follow-up (Day 4)	**Status**	Clinically stable. Noted some spontaneous respiratory effort, but likely insufficient for weaning. Improved level of consciousness when sedation lightened (GCS recorded as E4 V(T) M5 = GCS 9T). No desaturation, hypotension, or bradycardia reported.
	Ventilation	Airway patent via ETT. Still required ventilator support but degree of spontaneous effort noted.
	Hemodynamics	Stable (HR 100/min, BP 124/64 mmHg).
	Plan	**Transfer approved:** Patient deemed stable for transfer out of ICU to a lower dependency unit for continued monitoring, supportive care, and gradual ventilator weaning as neuromuscular function recovers.
	Treatments	Continuation of antibiotics, supportive care, and gradual reduction of respiratory support based on progress.
Post-ICU follow-up	**Neurology Recommendation**	Follow-up in 2 months.
		Prolonged recovery anticipated due to severe axonal polyneuropathy and phrenic nerve palsy, requiring potential long-term rehabilitation

The observation of paradoxical breathing upon the patient's admission to the emergency department is a cardinal clinical sign indicative of diaphragmatic weakness or paralysis. In normal respiration, the diaphragm contracts and descends during inhalation, causing the abdomen to expand. However, in cases of diaphragmatic paralysis, the diaphragm moves paradoxically, being pushed upwards by the abdominal contents during inspiration as the intercostal muscles attempt to draw air into the lungs. This paradoxical movement reflects a severe compromise in the patient's ability to ventilate effectively and was a critical indicator of the need for immediate ventilatory support. This clinical finding of diaphragmatic compromise was subsequently corroborated by the nerve conduction velocity (NCV) study, which definitively demonstrated bilateral phrenic nerve palsy. The phrenic nerves are responsible for innervating the diaphragm, and their paralysis results in the loss of diaphragmatic function, a primary driver of respiratory failure in these patients. The NCV and EMG studies further revealed a severe generalized axonal polyneuropathy, affecting both motor and sensory nerves. This electrodiagnostic finding is consistent with the profound and widespread damage inflicted upon the peripheral nerves by the β-bungarotoxins present in krait venom. These toxins exert their effects at the presynaptic terminal of the neuromuscular junction, where they irreversibly bind and disrupt the release of acetylcholine, the neurotransmitter crucial for muscle contraction. The resultant damage leads to functional denervation, characterized by muscle weakness and paralysis. The axonal nature of the polyneuropathy, as confirmed by electrodiagnostic studies, explains the often-protracted recovery period observed in these patients. Recovery hinges on the regeneration of damaged nerve axons and the formation of new neuromuscular junctions, processes that occur relatively slowly. This contrasts with conditions primarily affecting the myelin sheath of nerves, where recovery may be faster due to the potential for remyelination. While some *Bungarus* venoms contain postsynaptic α-neurotoxins, which act by binding to acetylcholine receptors on the muscle side of the neuromuscular junction, the predominant clinical picture and the electrodiagnostic findings in this case strongly suggest a primary and severe presynaptic effect. The rapid onset of paralysis, the severity of respiratory failure, and the characteristic axonal polyneuropathy are all more consistent with the action of β-bungarotoxins.[11-13]

A common challenge in the clinical management of snakebite envenomation is the difficulty in definitively identifying the species of the snake responsible for the bite. In many instances, as in this case, the snake is not captured, killed, or accurately described by the patient or witnesses. This lack of definitive identification necessitates a syndromic approach to treatment, wherein management decisions are guided by the observed clinical manifestations, such as neurotoxicity, coagulopathy (bleeding disorders), or local tissue necrosis. In this particular case, the rapid onset of severe neuromuscular paralysis strongly suggested envenomation by an elapid snake, a family known for its neurotoxic venoms. Within the elapid family, the *Bungarus* genus became the prime suspect, given the patient's presentation and the geographical context. In Indonesia, kraits are notorious for causing severe neurotoxic envenomation, and the local name "Weling" is commonly associated with these snakes. This reliance on the syndromic approach highlights the need for clinicians in snakebite-endemic regions to be well-versed in the typical clinical presentations associated with different groups of venomous snakes prevalent in their area. While this approach is often effective, it also underscores the limitations imposed by the lack of specific species identification.[14,15]

Standard laboratory tests play a crucial role in the overall assessment and management of snakebite patients. These tests are essential for evaluating the patient's general condition, assessing organ function, and detecting potential complications arising from the envenomation. In this case, laboratory investigations included serum electrolytes, complete blood count (CBC), coagulation studies, liver function tests, renal function tests, and arterial blood gas (ABG) analysis. While these tests are invaluable for guiding supportive care, they generally do not directly aid in diagnosing or predicting the severity of the neurotoxic effects of the

venom itself. Arterial blood gas (ABG) analysis is of particular importance in patients with respiratory compromise. It provides critical information about the patient's oxygenation status (PaO2), carbon dioxide elimination (PaCO2), and acid-base balance (pH, bicarbonate levels). In this case, ABG results were used to assess the severity of respiratory failure, guide ventilator settings, and monitor the patient's response to ventilatory support. Chest X-rays are also important, primarily for identifying pulmonary complications. In this patient, chest X-rays revealed bilateral pulmonary infiltrates suggestive of pneumonia, which was treated with antibiotics. Nerve conduction velocity (NCV) and electromyography (EMG) studies, while not typically performed as emergency procedures, provide invaluable diagnostic confirmation and prognostic information in cases of neuromuscular paralysis. These electrodiagnostic studies can help to differentiate between various causes of muscle weakness and paralysis, such as those originating from the central nervous system, the peripheral nerves, or the muscles themselves. In this case, the NCV/EMG studies definitively characterized the nature of the neuromuscular blockade, revealing severe generalized axonal polyneuropathy and bilateral phrenic nerve palsy. These findings not only supported the diagnosis of neurotoxic envenomation but also provided crucial prognostic information, indicating the likelihood of a prolonged recovery period. The development of rapid and accurate venom detection kits or biosensors holds the potential to significantly improve the diagnosis and management of snakebite envenomation. Such tools could facilitate rapid identification of the snake species involved, enabling clinicians to administer the most appropriate antivenom and tailor treatment strategies more effectively. However, it's important to acknowledge that these technologies are not yet widely available in many regions where snakebite is prevalent.[16-18]

In the management of severe neurotoxic snakebite, the prompt anticipation and effective management of respiratory failure is of paramount importance. This case underscores the critical need for rapid assessment and decisive intervention to secure the patient's airway and provide ventilatory support. The patient's presentation with dysarthria, paradoxical breathing, and hypoxia signaled impending respiratory failure and mandated immediate endotracheal intubation and mechanical ventilation. Any delay in establishing an adequate airway and providing ventilatory support in such cases can lead to severe consequences, including hypoxic brain injury and cardiac arrest. Once the patient is intubated, meticulous ventilator management within the intensive care unit (ICU) becomes crucial. In this case, the use of Pressure Support Synchronized Intermittent Mandatory Ventilation (PSIMV) mode with appropriate settings, including PEEP and FiO_2, provided effective ventilatory support, ensuring adequate oxygenation and carbon dioxide elimination. The importance of lung-protective ventilation strategies cannot be overemphasized, particularly in the context of concurrent pneumonia and acute respiratory distress syndrome (ARDS), as evidenced by the patient's P/F ratio of 139. Weaning patients from mechanical ventilation following *Bungarus* envenomation often presents a significant challenge and is typically a prolonged process. This is primarily attributed to the slow recovery of neuromuscular function due to the irreversible nature of presynaptic neuromuscular blockade caused by β-bungarotoxins. Patients may require mechanical ventilation for several days to weeks, depending on the severity of the envenomation and the extent of nerve damage. In this particular case, although the patient was transferred from the ICU after four days, while still likely dependent on ventilatory support, the NCV findings strongly suggested that the patient would require extended respiratory support beyond this initial period.During the period of mechanical ventilation, it is imperative to implement strategies to prevent ventilator-associated complications. These complications can include ventilator-associated pneumonia (VAP), which was already present or developed early in this case, barotrauma (lung injury caused by excessive pressure), and deconditioning (muscle weakness due to prolonged immobility).[19,20]

4. Conclusion

In conclusion, this case report elucidates the complexities of managing severe neurotoxic envenomation from a suspected Bungarus sp.,

highlighting the critical importance of prompt recognition and aggressive intervention to prevent fatal outcomes. The patient's rapid progression to respiratory failure underscores the potency of kraits' presynaptic neurotoxins and the consequent need for immediate airway management and mechanical ventilation. While antivenom remains a cornerstone of treatment, its limited efficacy in reversing established neuromuscular blockade emphasizes the indispensable role of prolonged ventilatory support and comprehensive intensive care. The electrodiagnostic findings of bilateral phrenic nerve palsy and severe axonal polyneuropathy not only confirmed the diagnosis but also indicated the potential for a protracted recovery phase, necessitating extended ventilatory support and rehabilitation. This case reinforces the need for heightened clinical vigilance and resource preparedness in regions where neurotoxic snakebites are prevalent. It also highlights the ongoing challenges in managing such cases, particularly in the absence of rapid and definitive diagnostic tools for snake species identification and the limitations of current antivenom therapy in reversing presynaptic neurotoxicity.

5. References

1. Kurniawan N, Muammar Kadafi A, Sih Kurnianto A, Ardiansyah F, Maharani T. Understanding snake bite cases pattern related to volcano-seismic activity: an evidence in Bondowoso, Indonesia. Biotropika. 2017; 5(3): 102–9.
2. Hadrani FH, Oktaputri A, Widodo G, Susetio Y. Sudden ocular complication after snake bite. Ophthalmologica Indonesiana. 2024; 49(S2).
3. Pothukuchi VK, Kumar A, Teja C, Verma A. A rare case series of Ischemic stroke following Russell's viper snake bite in India. Acta Med Indones. 2017; 49(4): 343–6.
4. Liwang F, Nuraeni F, Karyanti MR. Snake bite management in a toddler: a case report in Sumbawa Besar. Paediatr Indones. 2021; 61(4): 171–4.
5. Hermawati H, Mulyaningsih M, Widarti R. Increased knowledge about first treatment in snake bite cases for health cadres. Jurnal Penelitian dan Pengabdian Masyarakat Indonesia. 2024; 4(1): 892–9.
6. Weinstein SA, Jahan-Mahin MH, Baradaran M, Kazemi SM. Local envenoming by the eastern Montpellier snake, Malpolon insignitus fuscus (Fleischmann, 1831), Psammophiidae, and review of the evidence-based medical risks of bites by *Malpolon* spp. Toxicon. 2024; 248(107856): 107856.
7. Raut P, Raut S. Venomous snake bite in pregnancy in rural setting. Toxicon. 2024; 248(108005): 108005.
8. Sharma SK. Snake bite care in Nepal: Addressing the hidden crisis. Toxicon. 2024; 248(107915): 107915.
9. Dilip SG, Kanodia N, Raj P, Prasad Verma D. A case report on neurotoxic snake bite. Indian J Appl Res. 2024; 30–1.
10. Lucas GN. Snake bites in Sri Lankan children. Sri Lanka J Child Health. 2024; 53(3): 199–200.
11. Saini AMR. Neurotoxic snake bites in Saudi Arabia: Current status analysis. Ann Clin Anal Med. 2025; 16.
12. Rushton W, Ryan E, Rivera J, Kelly M, Atti S, Marshall S, et al. After the bite: evaluating a specialized clinic for follow-up care for snake envenomation. Clin Toxicol (Phila). 2025; 63(1): 16–22.
13. Xu S, Zhu L, Xuan J. Ultrasound evaluation of snake bites: recent developments and future directions. Med Ultrason. 2025.
14. Sreekumar A, Abraham SV, Rajeev PC, Chanchal A B V, Suseel A, Mathew D, et al. Educating healthcare workers in snakebite management: a study to determine the effectiveness of the snake bite life support workshop. Toxicon. 2025; 255(108235): 108235.
15. Vasconez-Gonzalez J, Delgado-Moreira K, Izquierdo-Condoy JS, de Lourdes Noboa-Lasso M, Gamez-Rivera E, Lopez-Molina MB, et al. Cerebrovascular events induced by venomous

snake bites: a systematic review. Heliyon. 2025; 11(4): e42779.

16. Kumar A, Banjara RA, Aneshwari RK, Khan J, Bernarde PS. A comprehensive review on recent advances in the use of ethnomedicinal plants and their metabolites in snake bite treatment. Front Pharmacol. 2025; 16: 1548929.
17. Kumar Thatoi P, Debasish Behera K, Mishra S, Panda B, Choudhury A, Barik P, et al. Clinical and laboratory parameters associated with acute kidney injury in patients with viperine snake bite: a prospective observational study in Central Odisha. J Neonatal Surg. 2025; 14(8S): 108–16.
18. Chauhan N, Thakur A, Raina SK. Preference for habitat contiguity: Geographical characterization of snake bite envenomation in Himachal Pradesh, India. Amrita J Med. 2025; 21(2): 88–90.
19. Mirakbari SM, Gorji R. Acute cerebral artery occlusion following Levantine viper (*Macrovipera lebetina*) bite: Gács sign in snakebite. Arch Iran Med. 2025; 28(4): 240–3.
20. Levine M, Ruha A-M, Wolk B, Caravati M, Brent J, Campleman S, et al. When it comes to snakebites, kids are little adults: a comparison of adults and children with rattlesnake bites. J Med Toxicol. 2020; 16(4): 444–51.