

Case Report

Bedside intubation with AMBU bag ventilation improves outcome for respiratory muscle paralysis in neurotoxic snake bite: a case report

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ABSTRACT

Neurotoxic snake envenomation can rapidly produce neuromuscular paralysis, bulbar dysfunction and life-threatening respiratory failure. Prompt airway protection, antivenom administration and ventilatory support are essential; however, mechanical ventilators are frequently unavailable in rural and resource-limited healthcare facilities. A 20-year-old man from rural Bangladesh presented approximately four and a half hours after a suspected Wall's krait bite to his right foot. His arrival was delayed by initial treatment from a traditional healer. On admission, he had altered consciousness, bilateral partial ptosis, excessive salivation and a positive broken-neck sign, indicating severe neurotoxicity. Although his initial oxygen saturation was 98% on room air, he rapidly developed worsening breathlessness, reduced consciousness, severe bradycardia and oxygen desaturation to 80%. Emergency bedside endotracheal intubation was performed, and ventilation was maintained manually using an AMBU bag. Neostigmine with atropine was administered, followed by three doses of polyvalent antivenom comprising a total of 30 vials. The patient was transferred approximately 55 km to a tertiary-care intensive care unit while continuous AMBU-bag ventilation was maintained. He subsequently received mechanical ventilation for approximately 72 hours. His neurological and respiratory functions gradually recovered, and he was successfully extubated on the fourth day. He remained clinically stable and was discharged without apparent neurological or respiratory complications. Immediate bedside intubation and continuous AMBU-bag ventilation can provide a lifesaving bridge to definitive mechanical ventilation in patients with neurotoxic snakebite-induced respiratory paralysis. Training healthcare professionals in manual ventilation and ensuring the availability of basic airway equipment may substantially reduce preventable snakebite mortality in resource-limited settings.

Keywords: Neurotoxic snakebite, Wall's krait, Respiratory muscle paralysis, AMBU-bag ventilation, Endotracheal intubation, Antivenom, Resource-limited setting

INTRODUCTION

Acute respiratory failure caused by neuromuscular paralysis is a major cause of morbidity and mortality in neurotoxic snake bite.^{1,2} Respiratory failure is a syndrome in which the respiratory system cannot perform the function of gas exchange, either oxygenation or elimination of carbon dioxide (CO₂). In this case of neurotoxic snake bite, hypercapnic respiratory failure occurred due to the effect of the snake's toxin, which attacked the neuromuscular junction, triggering acute

neuromuscular paralysis and making ventilation difficult. Neurotoxic envenomations have the potential to cause a broad spectrum of presentations, ranging from ptosis and ophthalmoplegia to respiratory arrest. Timely administration of anti-snake venom and ventilatory assistance can prevent mortality and morbidity in victims.³⁻⁵ The prevalence of respiratory failure is variable, ranging from 14% to 59.4%.^{6,7} Delayed arrival at hospital is an important contributing factor to fatal outcomes in such victims.⁵ In Bangladesh, many patients are treated by local traditional healers (Ojha), which leads to delayed

arrival at hospital, complicating early neuromuscular respiratory failure. Mechanical ventilatory support, along with anti-snake venom therapy, is the backbone of management in patients with respiratory failure due to severe neuromuscular snake envenomation.⁸ Immediate endotracheal intubation is necessary for airway protection and prevention of aspiration in patients with bulbar involvement.⁹ In our country, mechanical ventilatory support is not available in rural health facilities. If the patient can be intubated and ventilation maintained using an AMBU bag, the patient's chance of survival increases.¹⁰ AMBU-bag ventilation may therefore be a game changer in neuromuscular respiratory failure due to snake envenomation in remote health facilities.

CASE REPORT

A 20-year-old male, hailing from Sonarai, Domar, Nilphamari, Bangladesh, presented to the Emergency Department of the General Hospital, Nilphamari, on 29th July 2025 at 12:30 am, with a complaint of a snake bite to his right foot near the 2nd toe at 9:00 am on 29th July 2025 while he was working in a jute field, approximately 4.5 hours before examination (Figure 1). About half an hour after the bite, he developed pain at the bite site, severe headache and body aches. He ligated the right leg with a rope at three sites and visited the Domar Upazila Health Complex. As no snake antivenom was available at the health complex, he was referred to Nilphamari District Hospital. He was treated by a local traditional healer (Ojha), and the ligature was removed. As there was no improvement with this treatment, he was brought to our hospital.



Figure 1: Bite mark on the dorsum of the right foot near the 2nd toe at the time of examination.

On examination, the patient had altered consciousness; pulse 88/min, regular; blood pressure 120/80 mmHg; respiratory rate 25 breaths/min; oxygen saturation 98% on room air; spontaneous salivation; bilateral partial ptosis; and a positive broken-neck sign (Figure 2). Pupils were normal and reacting to light, and the chest was clear on auscultation. Local examination of the right foot showed mild swelling, bruising and clotted blood over the dorsum near the 2nd toe, without redness, warmth or tenderness. At this stage, the snakebite severity score was grade 3.⁸



Figure 2: Clinical signs at presentation: spontaneous salivation, bilateral partial ptosis and a positive broken-neck sign; pupils were normal and reacting to light, and the chest was clear on auscultation.

After clinical examination, blood samples were taken for a bedside 20-minute whole-blood clotting test, which was negative. While preparations for specific management were underway, the patient's breathlessness increased and his consciousness deteriorated over the next half hour. Oxygen saturation suddenly fell to 80%, and severe bradycardia (heart rate < 45 beats/min) developed; the snakebite severity score at this point was grade 9. The patient was intubated at the bedside with the assistance of an anesthesiologist (Figure 3), and ventilation was maintained with an AMBU bag (Figure 4). Injectable subcutaneous neostigmine 2.5 mg, along with intravenous atropine 1.2 mg, was administered for neurotoxic features before antivenom, as antivenom was not available in our store at that time. The first dose of polyvalent snake antivenom, containing 10 vials, was given by intravenous infusion, with bedside anti-anaphylactic measures ready, within 3 hours of hospitalization, following intravenous neostigmine and atropine. The patient was then referred to Rangpur Medical College Hospital, 55 km from Nilphamari, for ICU support, with ventilation maintained by AMBU bag throughout transfer.



Figure 3: Emergency bedside endotracheal intubation performed with the assistance of an anesthesiologist.



Figure 4: Ventilation maintained manually with an AMBU bag prior to and during transfer to the tertiary-care intensive care unit.

Table 1: Hematological and coagulation findings.

Investigation	Result
Haemoglobin	12.5 g/dl
Erythrocyte sedimentation rate	20 mm in the 1st hour
Total white blood cell count	$10 \times 10^9/l$
Neutrophils	68%
Platelet count	$251 \times 10^9/l$
20-minute whole-blood clotting test	Negative
Prothrombin time	16 seconds
Activated partial thromboplastin time	28 seconds

Table 2: Biochemical, cardiac and urinary findings.

Investigation	Result
Serum alanine aminotransferase	30 IU/l
Serum aspartate aminotransferase	45 IU/l
Serum creatinine	1.09 mg/dl
Cardiac troponin I	<0.01 ng/ml
Random blood glucose	110 mg/dl
Urine physical examination	Normal
Urine biochemical examination	Normal
Urine microscopic examination	Normal
Urine output	2400 ml/day

Investigations revealed a complete blood count showing hemoglobin 12.5 g/dl, erythrocyte sedimentation rate 20 mm in the first hour, total white blood cell count $10 \times 10^9/l$, neutrophils 68%, and platelet count $251 \times 10^9/l$ (Table 1). Liver function tests showed serum alanine aminotransferase 30 IU/l, serum aspartate aminotransferase 45 IU/l, prothrombin time 16 seconds, activated partial thromboplastin time 28 seconds, serum creatinine 1.09 mg/dL, serum cardiac troponin I <0.01 ng/mL, and random blood glucose 110 mg/dl. Urinalysis showed normal physical, biochemical and microscopic findings, with a urine output of 2400 ml/day (Table 2). The second and third doses of polyvalent snake antivenom, each containing 10 vials, were administered intravenously,

with bedside anti-anaphylactic measures ready, on the same day. The patient was maintained on mechanical ventilation for the next 72 hours.

On the 4th day, the patient regained full consciousness; pulse was 80/min, regular, and blood pressure 120/80 mmHg, and extubation was performed successfully. After extubation, oxygen saturation was 97% on 10 l/min oxygen, which was gradually reduced to 2 l/min over the next 48 hours, and thereafter he maintained saturations of 91-94% without supplemental oxygen. He was transferred from the ICU to the medicine ward with stable vital signs and remained clinically stable for a further 4 days before being discharged in a hemodynamically stable condition.

DISCUSSION

Neurotoxic snake bites are strongly associated with a high mortality rate due to rapid respiratory failure, particularly in rural regions.¹¹ In Bangladesh, 86% of victims first seek treatment from “Ojhas” (traditional healers/snake charmers), and only 3% consult a doctor first.¹² This delay in reaching hospital for early medical management often results in patients arriving with multiple complications, including respiratory failure.

Snake venom toxins cause two types of neuromuscular blockade: pre-synaptic and post-synaptic. Pre-synaptically active neurotoxins (beta-neurotoxins mostly neurotoxic phospholipase A2 toxins, PLA2s) bind to motor nerve terminals, leading to depletion of synaptic acetylcholine (ACh) vesicles, impaired ACh release, and subsequent degeneration of the motor nerve terminal. The binding of pre-synaptic toxins to the nerve terminal is irreversible.² Clinical recovery is slow, as it depends on regeneration of the nerve terminal and formation of a new neuromuscular junction; treatment with antivenom or acetylcholinesterase inhibitors is unlikely to be effective against pre-synaptic toxicity, and incomplete recovery with delayed effects is more likely. Pre-synaptic toxins are best exemplified by beta-bungarotoxin of kraits (*Bungarus* spp., especially Wall's krait). Post-synaptically active neurotoxins (alpha-neurotoxins) bind to post-synaptic muscle acetylcholine receptors.² Our patient was bitten by a Wall's krait, which contains beta-bungarotoxin, and required early emergency medical management with respiratory support. The snakebite severity score can be applied for early identification and prompt recognition of clinical severity to guide timely treatment; our patient's severity score rose from 3 to 9, prompting drastic measures.¹³

This patient presented with severe neurotoxic features and impending respiratory failure. He was managed with supportive measures, antivenom, and bedside ventilation maintained by AMBU bag, and was then transferred to the ICU, 55 km away, with continuous AMBU-bag ventilation. After 4 days of mechanical ventilation in the ICU and a further 6 days of medical treatment in the general ward, the patient recovered successfully and was discharged.

Early arrival at medical facilities, prompt diagnosis, and effective therapy for neurotoxic snake bites are crucial factors for favorable outcomes. Early initiation of antivenom, anticholinesterase therapy, and ventilator support is the recommended treatment protocol for venomous snakebite patients; these interventions are essential for preventing respiratory failure and saving the patient's life.¹⁴ Caring for critically ill patients in resource-limited settings remains challenging owing to the high burden of disease and high mortality from potentially treatable critical illnesses.¹⁵ Early initiation of bedside intubation with mechanical ventilation maintained by AMBU bag, together with antivenom and other supportive measures, can save the lives of patients with neurotoxic respiratory failure in resource-limited settings.¹⁰

CONCLUSION

In venomous snake bite with severe neurotoxic features, including respiratory failure, immediate medical management together with respiratory support via bedside mechanical ventilation can save many lives.

Recommendations

Rural and district-level healthcare facilities should ensure the availability of essential airway equipment, AMBU bags, emergency medicines and polyvalent antivenom, with personnel trained in early endotracheal intubation and manual manual ventilation. Community awareness and referral systems should be strengthened awareness and referral systems should be strengthened to reduce delays caused by traditional treatment and to ensure that patients with suspected neurotoxic snakebite receive prompt hospital-based assessment, antivenom therapy and respiratory support.

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