Case Report

# Neurotoxic snake bite with respiratory failure

**Prithwis Bhattacharya, Arpan Chakraborty**

**Abstract**

Thirteen patients with severe neuroparalytic snake envenomation admitted in intensive care unit with respiratory failure over a four months period. Initially ptosis and ophthalmoplegia, followed by bulbar palsy and respiratory muscle weakness was the common sequele. All of them received cardio-respiratory support with mechanical ventilation, anti-snake venom (median dose of 20 vials) and anticholinesterase therapy. Except one suffering from hypoxic brain injury due to delayed presentation, rest survived with complete neurological recovery. So good outcome in such cases is related with early cardio respiratory support and anti venom therapy

**Key words:** Anticholinesterase, envenomations, polyvalent anti-snake venom



## Introduction

Snakebites remain a public health problem in many countries even though it is difficult to be precise about the actual number of cases. According to toxicity, they are categorized as hematotoxic, neurotoxic and myotoxic. Among the neurotoxic group, the majority of bites are due to Naja naja (common cobra), Ophiphagus hannah (king cobra) and Bungarus caeruleus (Krait) in India. The snake venom consists of different enzymatic and nonenzymatic components loosely categorized as neurotoxins and hemorrhagens. In our ICU, most of the cases admitted are neurotoxic snakebites from the above-mentioned three commonest varieties. Neurotoxic envenomations have the potency to cause a broad spectrum of presentations starting from ptosis and ophthalmoplegia to respiratory arrest. Timely administered anti-snake venom and ventilatory assistance can prevent the mortality and morbidity of the victims.[1-5]

## Case History

In our ICU, all the patients (from eastern U.P) of neurotoxic envenomation presented with neuromuscular involvement with respiratory paralysis during the period of June to September in 2005. They were received in emergency OPD where they were resuscitated and shifted to ICU as early as possible. Detailed history and systemic examination, site of bite, any local reaction at the site of bite were recorded. Routine laboratory investigations including arterial blood gases and complete hemogram with coagulation profile were sent at the time of admission. APACHE - II scoring was done for every patient at the time of admission. All the patients were ventilated [Table 1] initially in controlled mode in Datex Ohmeda Centiva-5 ventilator. With the improvement of neuromuscular paralysis, weaning was implemented through SIMV-CPAP modes. If successfully tolerated, they were put on T-piece trial. Extubation was done after assessing clinically and arterial blood gas parameters [Table 2].

Polyvalent anti-snake venom (ASV) started as loading dose (50 ml over two hours) and maintenance infusion (50 ml six hourly). We have also used anticholinesterase (i.e. neostigmine started at a rate of 25 mcg/kg/hour) and anticholinergic (glycopyrolate) combination as infusion to

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**Table 1: Indications for mechanical ventilation**

| | |
|---|---|
| Acute ventilatory failure | pH <7.30, $PaCO_2$>50 mmHg, apnea |
| Impending ventilatory failure | |
| Tidal volume | < 3-5 ml/kg |
| Resp. rate and pattern | >25-30/min, labored and irregu lar pattern |
| Minute ventilation | >10 L/min |
| $PaCO_2$ trend | Increasing to over 50 mmHg |
| Vital signs | Increase in heart rate and blood pressure |
| Severe hypoxemia | $PaO_2$ - <60 mmHg at $FiO_2$ - >50% or $PaO_2$ - <40 mmHg at any $FiO_2$ Severe cyanosis |
| Prophylactic ventilatory support | To reduce pulmonary complications To avoid hypoxia |

**Table 2: Criteria for extubation**

| Criteria | Methods of assessment |
|---|---|
| Rapid breathing index | Respiratory rate/tidal volume in liter- <100 min/L |
| Blood gases | Acceptable blood gases on $FiO_2$ less than 40% and spontaneous minute ventilations less than 10 L/min $PaO_2/FiO_2$ more than 300 mmHg |
| Cardiopulmonary assessment | Absence of cardiopulmonary problems (e.g., CHF, pulmonary edema, pneumonia, tachycardia, arrhythmia, chest retractions, distended stomach.) |

reverse the neuromuscular blockade till ptosis improved in every case.[6] Bomb BS *et al*. in 1996 showed that anticholinesterases are the cornerstone of the neurotoxic bites, even in the absence of anti-snake venom. Outcome measure studied were survival, duration of ventilation and requirement of ASV and anticholinesterase.

Out of 13 patients [Table 3], neuroparalytic symptoms started within an hour of bite in eight individuals. Except in one, site of bite has been localized from history as well as bite mark. One patient has developed local cellulitis at the site of bite for which higher antibiotic coverage and low molecular weight dextran was given.

In all patients, anti-snake venom was given within a short period of the onset of symptoms and continued till significant recovery of power of all four limbs and intercostal muscles. ASV requirement was varying from10 vials to 30 vials maximum up to 48h of bite. No patient in our series demonstrated any hypersensitivity reaction to anti snake venom. Anticholinesterase therapy continued till the improvement of the ptosis [Figure 1].

In one case that presented with cardiac arrest and was revived after prolonged CPR, the anti-snake venom could be administered only 12h after the onset of symptoms. That patient had already aspirated before being brought to the hospital. His pulmonary condition deteriorated rapidly and he finally expired.

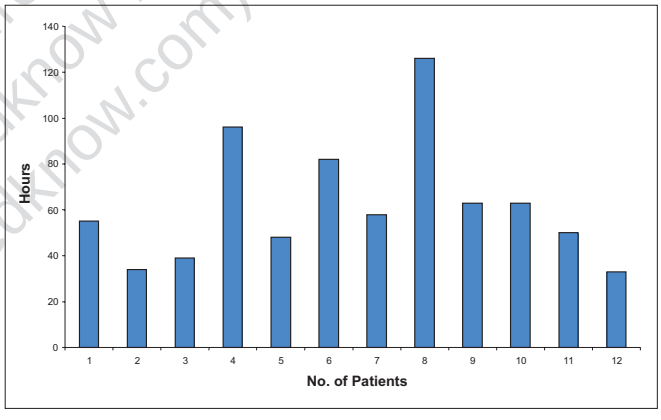


**Figure 1:** Duration of neostigmine therapy in survived patients



**Table 3: List of the patients**

| Age (yrs) | Sex | Site of bite | Type of snake | Onset of symptoms | APACHE - II | Treatment started after | Total ASV received | Duration of stay in ICU |
|---|---|---|---|---|---|---|---|---|
| 40 | F | Left hand | Cobra | 30 min | 9 | 5 hours | 20 vials | 82 hours |
| 30 | F | Left leg | Krait | 2 hours | 17 | 7 hours | 17 vials | 48 hours |
| 48 | M | Left leg | ? | 1 hour | 3 | 1 hour | 12 vials | 42 hours |
| 8 | F | Right leg | Cobra | Immediate | 17 | 1 hour | 13.5 vials | 5 days |
| 14 | M | ? | Cobra | Immediate | 17 | Immediately (Outside) | 27 vials | 60 hours |
| 35 | M | Right ear | ? | 2 hours | 17 | 4 hours | 25 vials | 96 hours |
| 20 | M | Lower extremity | Krait | 2 hours | 17 | 4 hours | 20 vials | 4 days |
| 20 | M | Lower limb | Krait | 3 hours | 19 | 4 hours | 30 vials | 150 hours |
| 10 | M | Left foot | Cobra | 1 hour | 19 | 4 hours | 28 vials | 72 hours |
| 38 | F | Right hand | ? Cobra | 1 hour | 17 | 12 hours | 20 vials | 88 hours |
| 23 | M | Scrotum | ? | 1 hour (paralysis started after 24 hours) | 8 | 24 hours | 12 vials | 72 hours |
| 45 | M | Right ear | ? | 3 hours | 5 | 8 hours | 13 vials | 35 hours |
| 10 | F | Left elbow | ? Cobra | 30 min | 17 | 2.5 hours | 10 vials | 67 hours (till death) |

APACHE II - Acute physiology and chronic health evaluation II, ICU - Intensive care unit

We have started ciprofloxacin and metronidazole as an empirical basis in all the patients on admission to prevent soft tissue infection at the site of bite. Most of them recovered uneventfully. In four patients with aspiration pneumonitis we had to add/change the antibiotics according to culture-sensitivity reports from endotracheal tube specimens.

No patients presented with any bleeding episode or renal involvement or any cardiac injury. No patient needed reintubation in our series

## Discussion

Ophitoxemia (clinical spectrum of venomous snake bite) starts with the onset of local changes within six to eight min of snakebite. Local pain, tenderness and reddish wheal followed by edema, swelling and appearance of bullae is a feature common to all snakes. Cobra and Kraits usually produce early wet gangrene whereas vipers produce a slower onset dry gangrene. Apart from fright and psychological shock, cobra bites can produce symptoms as early as 5 - 30 minutes or it may be delayed up to 10h. In one patient, neuroparalytic symptoms started after 24h of bite. They selectively produce a flaccid type neuromuscular paralysis. Muscles innervated by cranial nerves are involved earlier. While ptosis and ophthalmoplegia are among the first symptoms (found in all cases), the pupils and diaphragm are the most resistant to toxins. But pupillary changes (found dilated in three patients) can occur earlier as a consequence of respiratory arrest and hypoxia - as was evident in our cases. Cardiotoxic features include tachycardia, hypotension and ECG changes.

Pre-synaptic as well as postsynaptic neurotoxins of cobra and krait affect mainly muscles of eye, tongue, throat and chest causing respiratory failure.[7] Severity of envenomations and respiratory paralysis is related to dose of venom injected, potency of venom, anatomic location of bite, age, health and immune status of the victim and timely medical intervention.[8] In all patients timely administration of ASV and cardio respiratory support was led to favorable outcome.

Immediate endotracheal intubation is necessary in patients with bulbar involvement to protect airway. Weaning from mechanical ventilation is relatively easy as the patients are otherwise healthy and usually

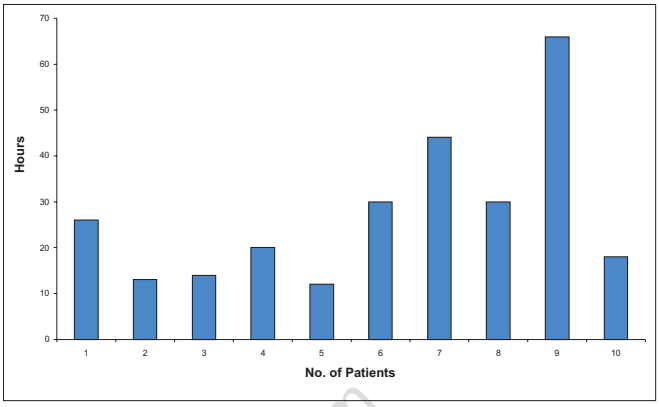


**Figure 2:** Duration of ventilatory support in survived patients

responsive to ASV within short period of time as in our study. Duration of mechanical ventilation in our patients is shown in [Figure 2].

In our study, we have used lower loading dose of ASV and maintenance infusion through syringe pump.[9] Anticholinesterase therapy was administered to all patients to combat postsynaptic toxins as most of them reached very early (median duration of four hours) to us.

## Conclusion

From our study we can conclude that administration of anti-snake venom with anticholinesterase therapy and cardio-respiratory support is the mainstay of therapy in neurotoxic envenomation with respiratory failure. Outcome is excellent if management is started early and before irreversible hypoxic insult.

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