

CASE REPORT

Neuroparalytic Snake Bite: A Rare Case Report and A Brief Review of Literature

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Introduction: Snake bite remains a public health problem in many countries. The potency to cause neurotoxic envenomations have a broad spectrum presentations which starts from ptosis and ophthalmoplegia & ends to respiratory arrest. Early diagnosis and management with anti-snake venom and ventilator support can prevent the mortality and morbidity of the victims.

Case report: A 39-year-old male presented with early morning with history of snake bite, deteriorate after 4 hours of admission. He improved with anti-snake venom and ventilator assistance. However, good outcome in such cases is related to early cardio-respiratory support and anti-venom therapy.

Conclusion: 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.

Keywords: Envenomation, Anticholinesterase, Anti-snake venom.

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INTRODUCTION

Snake bites 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 (common cobra), ophiophagusannah (kingcobra) and bungaruscaeruleus (krait) in india. The snake venom had different enzymatic and non-enzymatic components which

are divided into neurotoxins and hemorrhagens. The potency to cause neurotoxic envenomations have a broad spectrum presentations which starts from ptosis and ophthalmoplegia & ends to respiratory arrest. Early diagnosis and management with anti-snake venom and ventilator support can prevent the mortality and morbidity of the victims.^{1,2} Here, we report a rare case of neuroparalytic snake bite in which early diagnosis and management with ant-snake venom and ventilator support can prevent the mortality.

CASE REPORT

In our casualty a 39 years old male presented at early morning 2.30 am with history of snake bite on the back of right shoulder (fig :1) while sleep. On examination, his vitals were stable with a normal systemic examination. He was kept under observation in intensive care unit for close monitoring. He had no other significant past medical history. At around 6 am in the morning patient went into sudden bradycardia (35) with decreased respiratory efforts. Immediately the decision to intubate and ventilate is taken and ASV(anti-snake venom) started. Neostigmine was given. After 5 hours of admission, we had patient on ventilator, non sedated. On neurological examination findings were Unresponsive to stimulus, Pupils were dilated and fixed, Doll's eye absent, corneal & conjunctival reflex was also absent, No spontaneous breathing. The neurostatus remain same for that day and next day too. Neurophysician advised to continue same and CT Brain (plain) which was normal. EEG suggestive of normal activity. We continued same for one more day. Patient was given ASV (Polyvalent anti-snake venom started as loading dose (50 ml over two hours) and maintenance infusion (50 ml six hourly), neostigmine (started at the rate of 25 mcg/kg/hour),



Figure-1: Snake bite mark on back of right shoulder.

Low molecular weight heparin, enteral nutrition and supportive treatment. On 4th day patient started giving positive response in form of lid movements, flickering movements and minimal response to pain but pupil was still dilated and non responsive to light. On next day patient had significant improvement with obeying commands, muscle weakness (proximal > distal) and pupils were sluggishly reacting to light. While improving, the patient had sudden drop in saturation, tachycardia and tachypnea. In try of finding cause ECG, Chest X-ray and 2DEcho was done. 2DEcho was suggestive of RA & RV dilated, PASP 70 mm. Diagnosis of pulmonary thromboembolism made. Thrombolysis with recombinant tissue plasminogen activator (TNK-t-PA) 40mg bolus given and then kept on LMWH. Then repeat 2DEcho report revised suggestive of no residual effects. He was discharged after fifteen days from the day of admission. He was under regular follow-up and absolutely normal without any symptoms. Here, we present a case report of envenomation snake bite in which timely administered anti-snake venom and ventilatory assistance can prevent the mortality. However, good outcome in such cases is related to early cardio-pulmonary support and anti-venom therapy.

DISCUSSION

In developing countries, snake envenomation is a major cause of mortality and morbidity due to lack of advanced life support equipment. In India, snake bite envenomation is a common problem which is seen especially in the rural areas. There are around 2500 to 3000 species of snakes distributed worldwide and out of which 500 are venomous. Common species of snakes in India are the elapidae which consists of common cobra, king cobra and krait. Viperidae consists of Russell's viper, pit viper and saw-scaled viper. Around 45,900 deaths are estimated to result annually from snake bites in India.²

All over world around 2,000 species of snakes are known to us, of which 400 are poisonous. These snakes are belong to the families of elapidae, viperidae, hydrophidae, and colubridae. Every year, around 50,000 Indians die in 2, 50,000 incidents of snake bite, despite the fact that India is not home for the largest number of venomous snakes in the world, nor is there a shortage of anti-snake venom in the country. In 60%-70% of cases, snakebite occurs when the patients were asleep and site of bite is undetectable in 17% cases.³

Many patients with neurological manifestations presented without history of snake bites. Snake bites occurs because of neuromuscular paralysis of voluntary muscles which is caused by neuromuscular blockade. Irreversible binding of toxin to presynaptic portion makes clinical recovery slow in krait envenomation as recovery occurs only with the formation of new neuromuscular junctions. Pre-synaptic as well as postsynaptic neurotoxins of cobra and krait affect mainly muscles of eye, tongue, throat and chest causing respiratory failure. 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.⁴ Neurotoxins bind to acetylcholine receptors at the motor end-plate, where they first cause release of acetylcholine at the nerve endings at the myoneural junction and then damage the endings, preventing further release of transmitter. All this leads to a flaccid paralysis of the victim.

The common features of all snakes include local pain, tenderness, and reddish wheal followed by edema, swelling, and appearance of bullae. Early wet gangrene caused by cobra and kraits whereas vipers causes a slower onset dry gangrene. Most of the snakebites in our study were haematotoxic (60%), followed by neuroparalytic (40%) ones. Among the haematotoxic snake bites, bleeding from the site of the bite was the main manifestation, followed by cellulitis, haematuria and ecchymosis were observed in studies which were done in Maharashtra. The neuroparalytic symptoms which were seen, in the descending order, were ptosis, ophthalmoplegia, bulbar weakness, respiratory muscle involvement, and limb weakness.

The reported incidences of the neurological symptoms in 2 Sri Lankan studies on neuroparalytic snakebites were, ptosis in 70%–85% cases, respiratory muscle weakness in 18%–45% cases, ophthalmoplegia in 53%–75% cases, and limb weakness in 27%–54% cases respectively.⁵ According to a study by Saravu et al. which showed various symptoms of krait envenomation are abdominal pain, dyspnea, dysphagia, oliguria, respiratory paralysis, hypokalemia, coma, and renal failure. A study by Law et al. reported that case of krait envenomation which presented with coma and hypertension was discharged successfully.⁶ The study conducted by Trinh et al. on 1998 and 2007 showed manifestations of krait envenomation which consists of fang marks, numbness, bilateral ptosis, persistently dilated pupils, limb weakness, dyspnea, dysphagia, dysphonia, hyponatremia, rhabdomyolysis, and alternation in blood pressure.

Cardiotoxic features include tachycardia, hypotension and ECG changes. The time lag between bite and apnoea can be 30 min to 50 hrs. The apnoea can persist for 12 hours to 29 days. Structural damage to motor nerve terminal and motor axon follows rapidly and destruction of the nerve terminal is incomplete by 12 to 14 hours.

Other differential diagnoses such as myasthenic crisis, botulism, Guillain-Barre syndrome (Miller Fisher variant) were excluded based on history, clinical features, course of illness, and response to treatment.

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 responsive to ASV within short period of time. In a study by Prasarnpun S *et al.*⁷, 101 subjects of krait bite were studied. They were severely poisoned and required mechanical ventilation. The duration of mechanical ventilation ranged between 12 hours and 29

days. Neither polyvalent antivenom nor anticholinesterase as provided any significant benefit in reversing the neuromuscular paralysis or reducing the duration of ventilator support. The first aid consists of reassurance, immobilizing the bitten limb and transporting the patient to the nearest health care facility where definite treatment is provided. The things that are to be avoided includes application of tourniquet⁸; washing of the bite site with soap or any other solution to remove the venom; making cuts or incisions on or near the bitten area to remove the venom; use of electrical shock; freezing or applying the extreme cold to the area of bite; application of any kind of potentially harmful herbal or folk remedy; sucking out venom with mouth; giving the victim drink, alcohol or other drugs and also do not attempt to capture, handle or kill the snake, and patients should not be taken to quacks.

National Snakebite Management Protocol and WHO protocol have been formulated giving the standard management of snake bite. Early management consists of dealing with airway, breathing, and treatment of shock. Tetanus toxoid is administered if skin is breached. Cellulitis or local necrosis is treated by antibiotics. With oral paracetamol, pain can be relieved. Aspirin or non steroidal anti-inflammatory drugs should not be administered. ASV is available both in liquid and lyophilized forms and there is no evidence which proves superiority of one over the other. Lyophilized ASV, in powder form, has 5-year shelf life and requires only to be kept cool. ASV should be administered only when there are definite signs of envenomation which could be either coagulopathy or neurotoxicity. No role of local application of ASV is found as it may increase pain, swelling, and results in compartment syndrome.

Neostigmine is an anticholinesterase, which is particularly effective in postsynaptic neurotoxins such as those of the cobra and is not useful against presynaptic neurotoxin, that is, common krait and the Russell's viper¹⁰. After discharge from hospital, victim should be followed up and advised to return back for any worsening of symptoms like bleeding, pain or swelling at the site of bite, difficulty in breathing, altered sensorium, etc. The patients should also be explained about serum sickness which may manifest after 5-10 days.

CONCLUSION

From our case report 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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