# **Case Report**

# **Neurotoxic Snake Bite Poisoning**

<sup>1</sup>Ankita Kakkar, <sup>2</sup>Sushil Kumar

### Abstract

Snake bite is a significant health problem in India, particularly in the rural regions of the country. In general about 70% of bites are due to snakes which are not poisonous, of the rest, 15% are dry bites and only15% cause envenomation. Venom is the saliva of snake ejected during the act of biting, from the poison apparatus (the modified parotid glands). It can be, neurotoxic, vasculotoxic, or myotoxic in its action. Neurotoxicity is a key feature of some envenoming, and there are many unanswered questions regarding its manifestations. The polyvalent antisnake venom serum available in India is effective against most common poisonous snakes. Therefore, a prompt diagnosis and timely administration of polyvalent antisnake venom, in a case of snake bite can not only be life saving, but also prevent morbidity to a great extent. Neurotoxic snakes for example common krait hunt nocturnally, and are quick to bite people sleeping on the floor, often without waking their victims, since the venom is painless. Victims wake up later, paralyzed or die in their sleep.

In the present case report, we discuss the neurological manifestations, disease course and its outcome in one such patient of snake bite.

Key Words: Envenomation, Mortality, Morbidity, Manifestations

## Introduction:

Snake bite is a neglected tropical disease of global importance. [1] Data from the million deaths study in India estimates that snake bite deaths are more than 30 fold higher than recorded in official hospital returns. [2]

According to toxicity, they are categorized as haemotoxic, neurotoxic, and myotoxic. Among the neurotoxic groups, the majority of bites are due to Ophiphagus hannah (king cobra), Naja naja (common cobra), and Bungarus caeruleus (Krait) in India.

There are many challenges to the study of neurotoxicity after snake bite. There is considerable variation between individual patients in the clinical manifestations following envenoming by any particular species.

Clinical presentations of neurotoxicity are likely to be colored by the emotional response to a snake bite; other neurological changes are related to hypotension, shock and other organ dysfunction (such as renal impairment).

#### **Corresponding Author:**

<sup>1</sup>Assistant Professor Department of Forensic Medicine Rama Medical College, Mandhana Kanpur, U.P. E-mail: ankitatandon2002@yahoo.co.in <sup>2</sup>Associate Professor DOR: 21.04.2014 DOA: 29.05.14 Neurological manifestations of envenoming by the non-neurotoxic are such as those due to coagulopathy. Comparing findings from different studies is difficult as there is a lack of uniformity in description or grading of neuromuscular weakness, or in assessment of response to treatment.

Interpretation of neurophysiological findings is also difficult as different methodolics have been used between studies. [3]

However, timely administered antisnake venom and ventillatory assistance can prevent the mortality and morbidity of the victims.

### Case Report:

An 18 year old male, student presented to the emergency department of our hospital in the early hours of morning, with history of sudden onset difficulty in walking and difficulty in deglutition followed by unconsciousness of 4 hours duration.

He went off to sleep at night, but was woken up at dawn, as he was thirsty. He got up to fetch water from the kitchen, but experienced difficulty in walking. Further, when he tried to sip water, he found difficulty in drinking it too.

A few hours later, he suddenly became unconscious and was rushed to our hospital. His relatives gave past history of minor injury sustained in the left knee, seven days ago.

On general examination, patient was unconscious, unresponsive to deep painful stimuli with a normal pulse and blood pressure and depressed respiratory rate. Pallor, icterus, cyanosis clubbing, edema and lymphadenopathy were absent.

On systemic examination, cardiovascular respiratory and abdominal examinations were essentially normal. Central nervous system examination revealed generalized hypotonic but power could not be assessed, due to his unconsciousness.

All cranial nerves were normal. Light and superficial reflexes were present. Pupil was normal in size, with normal reaction.

Patient was mechanically ventilated because of poor respiratory efforts, and given antibiotics, along with anti tetanus serum (ATS) as there was previous history of injury sustained in left knee. Further, he was investigated for complete blood profile, CT scan head and CSF examination.

On 2nd day, the reports of all above investigation were normal. But the patient had not responded to the medications given to him.

On the 3rd day, he slightly regained consciousness, but was not well oriented.

A thorough physical examination was done. His neurological examination revealed bilateral drooping of eyelids, which suggested us that it could probably be a case of neurotoxic snake bite. Thereby we administered six vials of antisnake venom (ASV) diluted in 500 ml of normal saline to him, after sensitivity testing.

Another dose of antisnake venom was administered, in the form of four vials, after 6 hours, followed by four more vials after 12 hours. Myo-pyrolate 5ml diluted in 100 ml normal saline was also given slowly 8 hourly for three days. Next day, patient started showing improvement. Drooping of eyelids slowly reverted, and patient regained complete consciousness in one day's time.

On becoming conscious, he was able to recollect that while sleeping on the terrace at night, he was bitten near his right ankle by some insect (which he had not seen) .He ignored the bite and continued to sleep.

Fifth day, his respiratory efforts had improved considerably, so he was extubated. However recovery in muscular power of his lower limbs was not much satisfactory.

This patient of snake bite had fairly stable intensive care unit stay of seven days, after which he was discharged, and advised physiotherapy for the residual weakness in his lower limbs.

# Discussion:

Snake venom contains several types of polypeptide toxins, of which the neurotoxins

produce paralytic effect by binding to presynaptic and post synaptic sites at neuromuscular junction. [4]

Common neurological symptoms in decreasing order of frequency include ptosis (85.7%), ophthalmoplegia (75%), limb weakness (26.8%), respiratory failure (17.9%), palatal weakness (10.7%) and neck muscle weakness (7.1%). These are experienced usually within 6 hours of bite. (5) Following administration of antivenom, the signs of recovery become evident written a few hours to several days. [6]

However, in our patient, palatal weakness developed first, which was followed by weakness in lower limbs. Subsequently, he had difficulty in respiration, followed by drooping of both eyelids, a couple of days later.

Prompt recognition of envenomation and timely administration of antisnake venom (anti-sera) is a life saving measure and is the only effective treatment for neutralization of toxins that has entered the circulation. [7]

Polyvalent antivenom has no significant benefit in reversing respiratory paralysis and preventing delayed neurological complications. Polyvalent ASV is relatively safe, and allergic reactions after ASV injection can be prevented by premedication with adrenaline, IV hydrocortisone and antihistaminic. [8]

Anticholinesterases are beneficial against the postsynaptic toxins that induce myasthenia like block. [9] In animal models, subjected to high dose of snake venom, anticholinesterases have proven their efficacy as antidote in extending expected survival time. [10] However, despite their proven efficacy anticholinesterase, and antisnake venom forms mainstay of therapy, and dose up to 400 ml have been used. In our case, 14 vials of polyvalent antisnake venom were used.

Ventillatory support forms a cornerstone of envenomation therapy. Incidence of complication is directly proportional to the duration of venom in blood. Respiratory failure is the most common cause of mortality and morbidity in victims bitten by snakes. A mortality rate of 7.6% was observed in patients on intensive care management.

A prompt recognition of respiratory failure and timely mechanical ventilation can decrease morbidity and mortality .But due to poor availability at periphery and at larger district centre ASV, still remains mainstay of therapy. [8] **Conclusion:** 

It is concluded, that even in the absence history of snake bite, cases presenting with sudden onset of neurological symptoms, such as, weakness in limbs, respiratory paralysis and drooping of eyelids, a possibility of snake envenomation should be considered. Timely, administration of intravenous polyvalent ASV, .along with Ventillatory support proves to be life saving in all such cases.

#### **References:**

- Harrison RA, Hargreaves A, Wagstaff SC, Faragher B, Lalloo DG. Snake envenoming: a disease of poverty. Plos Negl Trop Dis 2009 (3): e569 DOI: 10.1371/journal. Pntd. 0000569.
- Mohapatra B, Warrell DA, Suraweera W, Bhatia P, Dhingra N, et al. Snakebite mortality in India: a Nationally representative mortality survey. Plos negl trop dis 2011 (5): e1018 DOI: 10. 1371/journal. Pntd. 0001018.
- Silva HJ. Neurotoxicity in Snakebite- The limits of our knowledge. Plos Negl Trop Dis 2013 ; 7(10): e2302. DOI: 10.1371/journal. pntd.0002302

- Warrel DA. Venomous snakes. In: Weatherall DJ, Ledinghan JGG, and Warrel DA, (eds) Oxford textbook of Medicine. 3<sup>rd</sup> ed. Oxford: Oxford University press; 1996: 1126-39.
- Kohli U, Sreedhar VK. Snake bite: An Unusual Cause of Acute Abdominal Pain. Indian Pediatrics 2007; 44:852-853.
- Seneviratne U, Dissanayake S. Neurological manifestations of snake bite in Sri Lanka. J Postgrad Med 2002; 48:275-278.
- Britt A, Burkhart K. Naja naja Cobra bite. Am J Emerg Med 1997; 15(5): 529-33.
- Bawaskar HS, Bawaskar PH, Punde DP, Inamdar MK, Dongare RB, Bhoite RR. Profile of Snakebite Envenoming in Rural Maharashtra, India. J Assoc. Phys. Ind 2008; 56:88-95.
- Akram S, Khurshid T. Successful revival of neurotoxic snake bite by artificial ventilation and cholinesterases. JCPSP 2000; 10: 267-9.
- Flachsenberger W, Mirtschin P. Anticholinesterases as antidote to envenomation of rats by the death adder (Acanthopis antarcticus). Toxicon 1994; 32(1):38-9.