

**A Case Report on Indian Common Krait Bite with Unusual Hypertension****Buchi Reddy Kunduru<sup>1\*</sup>, Hari hasan Chunchu<sup>2</sup>, Ramyakrishna Kalva<sup>3</sup>, Mahender Vattipelli<sup>4</sup>**<sup>1</sup>M.D. General Medicine, Rohini Super Specialty Hospital, Hanamkonda, Warangal, Telangana State, India<sup>2</sup>Pharm. D, Department of Pharmacy Practice, Rohini Super Specialty Hospital, and St. Peter's, Institute of Pharmaceutical Sciences, Hanamkonda, Warangal Telangana State, India<sup>3</sup>Pharm. D, Department of Pharmacy Practice, Rohini Super specialty Hospital, and St. Peter's Institute of Pharmaceutical Sciences, Hanamkonda, Warangal Telangana State, India<sup>4</sup>Associate Professor, Department of Pharmacy Practice, Rohini Super specialty Hospital and St. Peter's Institute of Pharmaceutical Sciences, Warangal, Telangana State, India**\*Corresponding author***Buchi Reddy Kunduru***Article History**

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**Abstract:** In India snakebite is an important cause of mortality and morbidity. Indian common krait (*Bungarus caeruleus*), the most poisonous snake and high mortality in the absence of signs of significant local envenoming. We present a case of 41 years' adult male admitted with common krait (*Bungarus caeruleus*) bite that remained in such a state for over 72 h before a gradual and sustained recovery from envenomation and its complication of hypertension by the use of Anti snake venom (ASV) and other supportive measures.**Keywords:** *Bungarus caeruleus*, Anti-snake venom, Hypertension.**INTRODUCTION**

In India snakebite is an important cause of mortality and morbidity [1]. According to the American Society of Tropical Medicine and Hygiene, more than 2.5 lakh cases of snakebites occur in India leading to 46,000 deaths every year. In India, two-third bites are by saw-scaled viper, about one-fourth by Russell viper, and a small proportion by cobra and krait [2]. Indian common krait (*Bungarus caeruleus*), the most poisonous snake well known for inflicting bites on sleeping people inside their homes and causing long-lasting severe neuromuscular paralysis with its venom being ten times more poisonous than cobras venom, and high mortality in the absence of signs of significant local envenoming [3,4].

We present a case of common krait (*Bungarus caeruleus*) bite that remained in such a state for over 72

h before a gradual and sustained recovery from envenomation and its complication of hypertension.

**Fig-1: Common krait (*Bungarus caeruleus*)****CASE REPORT**

A 41 years' adult male patient presented with the history of Snakebite-Krait (figure-1) on his right side of flank region with two fangs (figure-2b) while driving a tractor at his fields. Patient admitted after 26 hours of snake bite, on admission he had history of

ptosis (figure-2a), throat pain, and shortness of breath without any past medical history. Patient was a farmer and occasional alcoholic. On examination the patient was conscious, vitals are elevated blood pressure 160/120 mmHg with the pulse rate 94 b/min, respiratory

rate 24/min and bilateral pupils dilated i.e. 4.6 mm. Patient was advised with the chest X-ray, complete blood picture, Liver function tests (LFTs), serum electrolytes, random blood glucose found to be normal and Arterial blood gas (ABG) found to be abnormal (table-1). On admission patient was on ventilator support, Anti snake venom 1 vial IV 6<sup>th</sup>hourly(3000

units, 7.6 cc), Neostigmine 0.5mg IM 4<sup>th</sup> hourly, Atropine 0.6mg IV 4<sup>th</sup> hourly, Cefotaxime 1gm IV 12<sup>th</sup> hourly, Hydrocortisone 100 mg IV 8<sup>th</sup> hourly, Pantoprazole 40mg IV OD, Nifedipine 10mg 12<sup>th</sup>hourly, IV fluids infusion and monitor vitals 4<sup>th</sup> hourly.



**Fig-2: 2a-Ptosis, 2b- Fangs at right side of the flank region**

On day-2, Patient was conscious & coherent, complained of blurred vision, Palpitations, no bleeding, hematuria and blood pressure 160/110 mmHg. Patient was on ventilation support; vitals are monitored 4<sup>th</sup> hourly and continued the same treatment. On day-3, Patient was conscious & coherent, blood pressure 150/90 mmHg and no complaints, ventilation and continued the same treatment. On day-4, Patient was conscious & coherent, stable, pupils are normal and no

neurological deficit. Vitals are normal and discontinued the anti snake venom therapy. On day-5, Patient was normal, no ptosis, neurological deficit; vitals are normal and discontinued the Neostigmine, Atropine then continued the remaining treatment. From day-6 to day-9, patient was on observation and discharged with Patoprazole 40 mg OD, Nifedipine 10 mg OD, Multivitamin supplements.

**Table-1: Abnormal levels Arterial blood gas (ABG)**

| Parameter        | Results        |
|------------------|----------------|
| Na+              | 130.6 mmol/L ↓ |
| K+               | 4.17 mmol/L    |
| Cl               | 95.6 mmol/L ↓  |
| Hct              | 54.1 %         |
| pH               | 7.4            |
| pCO <sub>2</sub> | 38.1 mmHg      |
| pO <sub>2</sub>  | 289.5 mmHg ↑   |
| HCO <sub>3</sub> | 25.2 mmol/L    |

**DISCUSSION**

Common krait (*Bungarus caeruleus*) is distributed throughout South Asia, and is responsible for large numbers of cases of severe neurotoxic envenoming each year [5]. 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%) and ischemic stroke [6, 7]. These are experienced usually within 6 hours of the bite. In this case, patient admitted with common neurological symptoms of ptosis, throat pain, respiratory failure, mainly these symptoms are developed within 6 hours of snake bite but this patient shows these symptoms after 20-24 hours.

Disturbance in relation to krait bite characterized by hypertension, tachycardia and mydriasis has been well described in the literature [8]. Typical of krait bites, local symptoms were minimal or absent in all cases. The autonomic abnormalities are thought to result both from the action of the pre-synaptic neurotoxin which causes reduced parasympathetic activity, as well as by producing blockade of receptor sites such as presynaptic-2 adrenoceptors, thereby inhibiting the inhibition of neutrally mediated release of norepinephrine with resultant sympathetic hyper-reactivity [9]. In this case, patient presented with the elevated/accelerated blood pressure of 160/120 mmHg, 94 b/min of pulse rate and bilateral pupils dilatation. Rare findings of elevated blood pressure were observed in this patient and treated with Nifedipine 10mg 12<sup>th</sup> hourly.

Patient was on Antivenom Therapy and it was the mainstay of treatment for krait envenomation. Many of the symptoms are ameliorated or entirely eliminated by the antivenom alone. Severe muscle paralysis developed by the patient and persists, treated with 0.6 mg of Atropine IV 4<sup>th</sup> hourly. Followed by giving 0.5 mg of Neostigmine IV 4<sup>th</sup> hourly. 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 viper [10].

## CONCLUSION

Progressive paralysis along with ophthalmic manifestations of neurotoxin is seen commonly in victims of snake bite. However, ASV, 72 hours' ventilation and other supportive measures were taken to reduce morbidity and hypertension is unusual neurological complication this labile hypertension was treated with Nifedipine in this case of common krait envenomation.

## REFERENCES

1. Warrell DA. The clinical management of snake bites in the Southeast Asian region. *Southeast Asian J Trop Med Public Health* 1999; 30:1-84.
2. Singh M. *Medical Emergencies in Children*, 5th ed. New Delhi: Sagar Publications, 2012; 754–755.
3. Lalitha V, Pillai, Dhananjay Ambike, Saifuddin Husainy, Anil khair, Ashok Captain, Ulrich Kuch. Severe neurotoxic envenoming and cardiac complications after the bite of a Sind Krait<sup>®</sup> (*Bungarus cf. sindanus*) in Maharashtra, India. *Trop Med Health*. 2012; 40(3):103-8.
4. Agrawal PN, Aggarwal AN, Gupta D, Behera D, Prabhakar S, Jindal SK. Management of respiratory failure in severe neuromuscular snake envenomation. *Neurol India*. 2001; 49:25-8.
5. Valenta J. *Venomous snakes—Envenoming, Therapy*. 2nd Edition. New York: Nova Science Publishers, Inc.; 2010.
6. Narang SK, Paleti S, AzeezAsad MA. Acute ischemic infarct in the middle cerebral artery territory following a russell's viper bite. *Neurol India* 2009;57:479-80.
7. Boviatis EJ, KouyialisAT, Papatheodorou G. Multiple hemorrhagic brain infarcts after viper envenomation. *Am J Trop Med Hyg*2003;68:253-7.
8. Kularatne SA. Common krait (*Bungarus caeruleus*) bite in Anuradhapura, Sri Lanka: A prospective clinical study, 1996-98. *Postgrad Med J*. 2002;78:276–80.
9. Laothong C, Sitprija V. Decreased parasympathetic activities in Malayan krait (*Bungaruscandidus*) envenoming. *Toxicon*. 2001; 39:1353–7.
10. Anil A, Singh S, Bhalla A, Sharma N, Agarwal R, Simpson ID. Role of neostigmine and polyvalent antivenom in Indian common krait (*Bungarus caeruleus*) bite. *J Infect Public Health* 2010;3:83-7.