http://jmscr.igmpublication.org/home/
ISSN (e)-2347-176x ISSN (p) 2455-0450
crossref DOI: https://dx.doi.org/10.18535/jmscr/v7i12.90

IGM Publication
Journal Of Medical Science And Clinical Research
An Official Publication Of IGM Publication

A Case of Krait Snake Bite Responding to Calcium Gluconate Therapy

Authors
Dr B. Sreedevi[1*], Dr P.V.V. Satyanarayana[2], Dr P.Satish Srinivas[3]
[1, 2,3]Department of General Medicine, Rangaraya Medical College, India
*Corresponding Author
Dr B. Sreedevi
Department of General Medicine, Rangaraya Medical College, India

Abstract
Neuroparalytic snake bite is a common emergency encountered in India. Common krait (Bungarus Caeruleus) and cobra (Naja Naja) are important snakes causing neuroparalysis. Kraits belong to Elapidae family. Envenomation by elapids usually causes neurotoxic manifestations. Krait bite doesn't usually cause local signs & renal complications but may cause hematological abnormalities. Acute neuromuscular weakness with respiratory involvement is the most clinically significant neurotoxic effect. Neurotoxic manifestations in Krait bite are due to presynaptic inhibition, where calcium acts as a neurotransmitter; as a result, neurotoxic manifestations doesn't respond to Atropine neostigmine therapy but can respond to Injection Calcium gluconate therapy.
Keywords: *Krait, Elapidae, Neuroparalytic, Presynaptic inhibition, Injection Calcium gluconate.*

Introduction

Kraits (Bungarus species) belong to the Elapidae family. Neurotoxicity is a well-known feature of envenoming due to elapids. Krait bite doesn't usually cause local signs & renal complications but may cause hematological abnormalities. Neurotoxic manifestations include- vomiting, headache, giddiness, weakness, & lethargy in the preparalytic stage and ptosis, ophthalmoplegia, drowsiness, convulsions, bulbar paralysis, respiratory failure, & death in paralytic stage. Neurotoxic manifestations in krait bite are due to presynaptic toxicity.

Case Report

A 15-year-old male patient was brought to the emergency department with a history of being bitten by a Krait snake on index finger of left hand two hours back, presented with chief complaints of drooping of eyelids, breathlessness, and increased salivation. O/e: Bilateral ptosis present, RR:40/minute, SBC:10, SPO_2:70% @room air, PR : 80/minute, BP: 130/80 mmHg. The patient was intubated in view of respiratory paralysis and put on the mechanical ventilator. l/e : two fang marks with no local signs of inflammation. **Investigations:** 20-minute WBCT: clotted, complete blood picture, PT, INR, RBS, RFT, LFT, ECG are within normal limits. ABG revealed hypoxemia. **Treatment given:** Inj ASV was given as per the schedule Atropine neostigmine (AN) trail done after administration of the first dose of ASV. In AN trial 0.6ml atropine given intravenously, then 1.5mg neostigmine is administered intramuscularly, repeated after 30 minutes.

The patient was observed for 1 hour. No response to Atropine neostigmine trial. Injection calcium

gluconate 10cc given IV slowly over 5-10 minutes, every 6th hourly. 50% improvement in ptosis after the second dose was observed and complete recovery on 3rd day, and weaned off from ventilator on the 4th day. Supportive therapy in the form of antibiotic therapy, fluid therapy, ET care was given. The patient developed dysphonia on the 5th day, which improved with steroid therapy.

Discussion

Neurotoxic signs of envenomation like ptosis improved as early as 30 minutes after ASV administration in case of cobra bite (postsynaptic toxicity), but it takes a considerable amount of time in case of Krait bite (presynaptic toxicity). The bungarotoxins present in Krait venom have phospholipase A2 activity and hydrolyze phosphoglycerides, thereby producing neuromuscular blockade by inhibiting the release of acetylcholine from the presynaptic membrane[1,2]. Krait venom (Beta-bungarotoxin) usually causes presynaptic toxicity where calcium acts as a neurotransmitter; as a result, neurotoxic manifestations doesn't respond to Atropine neostigmine[3] therapy but can respond to Inj calcium gluconate[4] therapy. Regeneration of presynaptic receptors is a natural process and may take 4-5 days to regenerate. Despite severe neurotoxicity, patients who receive antivenom & ventilatory support along with calcium gluconate therapy in time recover early and completely.

Conclusion

Krait bite with neuroparalytic manifestations may respond to Inj calcium gluconate therapy along with ASV, as Krait venom (beta-bungarotoxin) usually causes presynaptic inhibition where calcium acts as a neurotransmitter.

References

1. Singh G, Pannu HS, Chawla PS, Malhotra S. Neuromuscular transmission failure due to common Krait (Bungarus caeruleus) envenomation. Muscle Nerve. 1999: 22:1637-43.
2. Lewis RL, Gutmann L.Snake venoms and the neuromuscular junction. Semin Neurol. 2004:24:175-9.
3. 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.
4. KK Pareek. Medicine update 2019(volume 2): Management of snake bite: 225-1120.