

Occult Snake Bite: A Neurological Dilemma

Emmanuel Paul¹, Mahesh Chavan², Hansraj Kamble³

Vilasrao Deshmukh Government Medical College, Latur, Maharashtra, India

Abstract: ***Introduction:** Occult snakebites, wherein patients are unaware of the bite and fang marks are absent, have been reported in kraits, an endemic neurotoxic snake belonging to the Elapidae family. Owing to a lack of snake bite history and unavailability of specific diagnostic tests, severe envenomation presents a challenge for physicians, unless they are aware of it and a high level of suspicion is maintained. **Case report:** A 17 years old male presented with complaints of sudden onset of both lower limb weakness, pain in both legs and low back pain of 1 day duration. Blood investigations done including complete blood count, liver and renal function tests, serum electrolytes were within normal limit. Csf study and MRI brain with whole spine screening were done and were within normal limit. Nerve conduction study was also done and was within normal limit. Patient was suspected to be having occult snake bite and was given Antisnake venom, Inj. Neostigmine along with Inj. Atropine and patient was drastically improved. **Discussion:** Krait bites are sometimes painless and occur during the night. Often, a history of snakebite and visible fang marks are absent. Patients usually experience symptoms like abdominal pain, nausea, vomiting, and malaise. This is followed by ptosis, external ophthalmoplegia, distal muscle weakness, and lastly diaphragmatic and respiratory muscle involvement. Respiratory failure is the most common cause of death. Autonomic dysfunction leads to internal ophthalmoplegia thus mimicking brain death. **Conclusion:** Occult snake bite Owing to a lack of snakebite history and unavailability of specific diagnostic tests, the diagnosis presents a challenge for practicing physicians, unless they maintain a high degree of suspicion.*

Keywords: Occult bite, Krait, ASV, Neostigmine

1. Introduction

Neurotoxic snakebites, a common emergency in tropical countries, may manifest from mild ptosis and ophthalmoplegia to severe flaccid paralysis and respiratory failure⁽¹⁾. More severe neuromuscular paralysis has also been reported, labeled variously as locked-in syndrome, early morning neuroparalytic syndrome, or “brain dead” presentation. Although a majority of these cases are diagnosed by a history of snakebite or the presence of visible fang marks, occult snakebites, wherein patients are unaware of the bite and fang marks are absent, are an underreported entity leading to a lack of knowledge about its existence. This has been described in kraits – an endemic neurotoxic snake belonging to the Elapidae family. They are believed to be nocturnal and possess small teeth. As a result, the victim is often unaware of the bite, and fang marks are invisible. As primary care physicians are usually the first respondents to such patients, it is essential that they are aware of this entity to prevent misdiagnosis and delayed treatment. Owing to a lack of snakebite history and unavailability of specific diagnostic tests, this diagnosis presents a challenge for practicing physicians, unless they maintain a high degree of suspicion.

2. Case Report

A 17 years old male presented with complaints of sudden onset of both lower limb weakness, pain in both legs and low back pain of 1 day duration. No history of altered sensorium, visual disturbances, difficulty in hearing, difficulty in swallowing, difficulty in speech, numbness of limbs.

No relevant past medical and family history.

Patient was initially evaluated at casualty where vitals were within normal limit, power of lower limbs were of grade 3 and plantar reflex were mute. Other neurological

examination were within normal limit. Patient was shifted into intensive care unit and were investigated.

Blood investigations done including complete blood count, liver and renal function tests, serum electrolytes and were within normal limit. Csf study and MRI brain with whole spine screening were done and were within normal limit. Nerve conduction study was also done and was within normal limit.

On next day morning patient developed drooping of both eyelid and weakness of both upper limb. On examination bilateral ptosis were present and power of upper limbs was grade 2.

Patient was suspected to be having occult snake bite and was given 10 vials of ASV, Inj. Neostigmine 1.5mg iv stat f/b 0.5mg iv 5 doses at gap of half hour interval, f/b 1mg every hour until ptosis resolved along with 0.6mg iv atropine. Following total dose of 20 vials of ASV and total of 8mg of neostigmine patient was drastically improved. On next day patient was shifted to general ward and then discharged.

3. Discussion

Krait bites are sometimes painless and occur during the night. Often, a history of snakebite and visible fang marks are absent. Neuroparalytic snakebite patients present with typical symptoms within 30 min– 6 hours in case of Cobra bite and 6 – 24 hours for Krait bite; however, ptosis in Krait bite have been recorded as late as 36 hours after hospitalization. These symptoms can be remembered as 5 Ds and 2 Ps. 5 Ds – dyspnea, dysphonia, dysarthria, diplopia, dysphagia. 2 Ps – ptosis, paralysis⁽²⁾. All these symptoms are related to 3rd, 4th, 6th and lower cranial nerve paralysis. Finally, paralysis of intercostal and skeletal muscles occurs in descending manner. Other signs of impending respiratory failure are diminished or absent deep tendon reflexes and head lag. Additional features like stridor, ataxia may also be

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seen. Associated hypertension and tachycardia may be present due to hypoxia.

The venom of kraits contains three major types of neurotoxin. α - Bungarotoxins cause a failure of neuromuscular transmission by binding to post - synaptic nAChR at the neuromuscular junction (NMJ). Similar toxins are found in the venoms of all elapid snakes and their close relatives, the sea snakes. κ - Bungarotoxins are found exclusively in the venom of kraits. They are structurally similar to the α - bungarotoxins, bind to neuronal nAChR but are minor components of the venom⁽³⁾. The β - bungarotoxins constitute >20% of the protein content of the venom and are the most toxic components of the venom. They are pre - synaptically active neurotoxic phospholipases A₂. Exposure to these toxins *in vivo* and *in vitro* causes the failure of neuromuscular transmission for 2–3 h, and the depletion of synaptic vesicles from nerve terminal boutons is a primary pathological feature of toxicity. Structural damage to the motor nerve terminal and terminal components of the motor axon follows rapidly and destruction of the nerve terminal is complete by 12–24 h. It has been suggested that β - bungarotoxin (alone or in combination with α - bungarotoxin) is primarily responsible for the severe paralysis associated with envenoming bites by kraits. The underlying hypothesis is that the onset of paralysis is caused by the depletion of synaptic vesicles from the nerve terminal, the destruction of the terminal boutons explains the phase of profound treatment - resistant paralysis and the slow recovery of neuromuscular function reflects the regeneration of nerve terminals and the re - innervation of the denervated muscle fibres

In summary, the above cases highlight the important role of primary care physicians in recognizing occult dramatic neurotoxic snakebites and subsequent severe neuromuscular paralysis as an important treatable medical emergency. In patients presenting with unexplained neuromuscular paralysis, especially with onset early in the morning, it is important to consider occult krait bite.

4. Conclusion

Occult snake bite owing to a lack of snakebite history and unavailability of specific diagnostic tests, the diagnosis presents a challenge for practicing physicians, unless they maintain a high degree of suspicion.

References

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