Case Report

A clinical dilemma in an unconscious patient

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Abstract

Snake bite is a major life-threatening emergency seen more commonly in rural tropical countries. In general, about 70% of the bites are nonpoisonous, 15% are dry bites, and only 15% cause envenomation. Venom is the saliva of snake ejected during biting, from the poison apparatus (the modified parotid glands). It can be neurotoxic, vasculotoxic, or myotoxic in its action. The polyvalent antisnake venom is effective against most common poisonous snakes. Prompt diagnosis and timely administration of the polyvalent antisnake venom can reduce mortality and morbidity to a great extent. We present a case which was brought to the emergency department of without any previous forthcoming history of snakebite, with symptom such as abdominal pain, chest pain, vomiting, and respiratory distress followed by loss of consciousness. The patient was timely resuscitated and with prompt use of polyvalent ASV and neostigmine the patient recovered without any neurological symptoms within a week.

Keywords: Envenomation, ptosis, pulmonary edema, snake bite

Introduction

Every year, 49,000 people in rural India die from snakebite poisoning.[1] Data from the million death study in India estimates that snake bite deaths are more than 30 fold higher than recorded in the official hospital returns.[2] In 2017, WHO categorized snakebite as a high-priority neglected tropical disease.[3]

Snake bites can happen at home by cobras dwelling on roof top and by kraits which nocturnally enter homes in search of prey like mice or lizards. It is a well-established fact that every species can produce a myriads of manifestations.[4] Neurotoxic snake bites, especially krait can present with painless bite without local inflammatory signs.[5]

Case Report

A 49-year old female housewife was brought to the Emergency Department of Tata Main Hospital on an early morning with history of sudden loss of consciousness for 1 hour. She went to bed at 9.30 pm the previous night, and started complaining of abdominal pain, chest pain, and vomiting since 12 AM, uneasiness and respiratory distress at 3 AM, and being unresponsive since 4 AM. She was brought to the Emergency department in an unconscious state after being referred from a primary care centre. There was no history of trauma, animal bite, injections, or drug abuse. There was no past history of diabetes, hypertension, CVA, IHD, or seizure disorder.

On examination, the patient was unconscious, GCS- 6/15, mild pallor, icterus, cyanosis, clubbing, edema, and lymphadenopathy were absent. Blood pressure was 180/100 mm Hg in right arm supine position, pulse was 120/min, regular, good volume, SpO2 70% on air. There were no rashes or any marks of insect bite or any fang mark.

On systemic examination, bilateral basal crepetations present in chest, S1, S2 heart sound heard, no murmer was audible. Abdomen was soft, no tenderness, and no organomegaly was observed. Examination of the central nervous system revealed hypotonia but power could not be assessed due to unconsciousness. Superficial and deep reflexes were present.

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Pupils were bilaterally dilated and poorly reacting to light. Neck was soft and no meningeal signs were present. Plantar response was flexor bilaterally.

The blood investigations in the Emergency department showed RBS-247 mg/dl, Parachek- negative, ABG showed pH-7.25, K-2.3, Na-130, pCO2-26, HCO3-13, lac-3.9. ECG showed sinus tachycardia and Trop T was negative.

The patient was treated in the emergency department with oxygen at 5 L, IV access, injection pantoprazole 40 mg iv and injection ondansetron 8 mg IV stat given. The patient was intubated and gastric lavage was performed; IV fluids 500 ML of DNS with 40 meq KCL @ 10 meq/hour was started, injection tetanus toxoid 0.5 cc im and injection ceftriaxone 1 gm IV stat. The patient was shifted to ICU with a differential diagnosis of (?) CVA, (?) unknown poisoning, (?) aspiration pneumonia, (?) MI/LVF, (?) and snake bite.

Radiological investigations done in emergency showed pulmonary edema (L >R) in X-ray and CT of brain show no hemorrhage/infarct.

In ICU, the patient was under ventilator support, PCV, FiO2-0.3. Injection Frusemide 20 mg IV BD, antibiotics Piperacillin + Tazobactum 4.5 gm iv TDS, and NTG drip at 0.6 ml/hour was started. ASV 10 vials in 5% dextrose iv over 1 hour, Atropine 2 amp iv over 12 hours infusion, Neostigmine 2.5 mg over 12 hours, Pantoprazole 40 mg iv BD, and Ondansetron 8 mg iv TDS were started.

By evening, the patient's condition was GCS 6/15, BP– 160/110 mm of Hg, SpO_2 is 100% on mechanical ventilator, P/R-103/min, low volume, RR- 18/min, Chest- B/L fine basal crepitations, CVS- S1S2+, and CNS bilateral pupils dilated poorly reacting to light.

On the second day, the patient's condition was GCS- 9/15 on ventilator, BP- 160/100 mm Hg, pulse- 110/min, chest- B/L fine basal creptations+, CVS- S1S2+, CNS- B/L pupils dilated, reacting to light, ptosis + and bilateral plantar flexor. The patient was changed to CPAP Mode, connected to T piece. 15 vials IV ASV was given while rest treatment remained the same.

On the third day, the patient's GCS improved to 10/15, BP- 160/120 mm Hg, pulse- 130/min, chest- B/L VBS+, CVS- S1S2+, CNS- ptosis +, bilateral pupils dilated, bilateral plantar flexor. X-ray finding showed cleared pulmonary edema. The patient was extubated, Tab Carvedilol 3.125 mg BD was started, Inj Atropine 1 amp q8h, Inj Neostigmine continued at 3 ml/hour and rest treatment continues the same.

On the fourth day, the patient improved with GCS- 15/15, BP- 130/70 mm hg, pulse- 96/min, Spo2- 100% with 5 litres of O2, Chest- B/L VBS+, CVS- S1S2+, CNS- no ptosis, no neurological deficit. The patient was transferred to ward and neostigmine and atropine were stopped.

On the fifth day, she was conscious, oriented, pulse- 84/min, BP- 130/80 mm hg, Chest- B/L VBS +, CVS- NAD, CNS- no neurological deficit. Finally, she was discharged after six days of hospital stay with a final diagnosis of neurotoxic snake bite.

Discussion

Snake venom contains several types of polypeptide toxins of which the neurotoxins produce paralytic effect by binding to presynaptic and postsynaptic sites at the neuromuscular junction.[6] Studies have shown that common neurological manifestations are ptosis (85.7%), followed by opthalmoplegia (75%), limb weakness (26.8%), respiratory weakness (17.9%), palatal weakness (10.7%), neck muscle weakness (7.1%), and delayed sensory neuropathy (1.8%).[7] These are experienced within six hours of the bite. Following administration of antivenom, the signs of recovery become evident within a few hours to several days and the duration of complete recovery ranges from four hours to two weeks.[7]

Common neurotoxic snakes in India includes Cobra (*Naja naja*) and Krait (*Bungarus caeruleus*). Abdominal pain, the cardinal feature of krait bite can precede the neurological symptoms by several hours. Krait bite are commonly reported between 2–3 am and those sleeping on floor are at greater risk. Painless bite with paucity of local tissue reaction is a typical feature of Krait bite.[8] ASV is most effective when administered within few hours of krait bite; hence, high degree of suspicion is required as the bite is not frequently witnessed and the initial symptoms can be non-neurological.

Studies have shown that we may not find bite mark on exposed part of skin and, in that case, other unusual sites like scalp, or other hairy areas should be examined after shaving it off. Long chain toxin such as beta bungarotoxin in krait venom is found to be similar to botulinum toxin and preservation of deep tendon reflexes in botulism can be the differentiating features from krait bite.

In our patient, there was no history of snake bite and no signs of any inflammatory response were present. Abdominal pain, vomiting, and chest pain without any neurological manifestation were the initial symptoms in our patient. The patient presented to ED in an unconscious state; so, proper neurological evaluations for ptosis could not be done. As rapid venom antigen detection like ELISA was unavailable in our setup, ASV was started even though classical features were absent. The patient started responding. Once the patient's condition improved after 24 hours and she became conscious, ptosis became evident and it further supported the diagnosis of neurotoxic snake bite.

In case of neurotoxic envenomation with respiratory distress and neuromuscular paralysis, antisnake venom, anticholinesterase therapy, and cardiorespiratory support remains the mainstray

of treatment.[9] Anticholinestrase (Neostigmine) prevents the degradation of acetylcholine—which can reverse respiratory failure and neurotoxic symptoms (postsynaptic). Acute dyspnea without wheezing is also a well-characterized feature of progressive neurotoxic envenoming (leading to insufficient respiratory muscle strength and increasing Neurological symptoms) which requires rapid treatment with antivenom +/- assisted ventilation.[10]

Conclusion

We report this case because no suggestive history or telltale signs were present. The patient had a history of abdominal pain, chest pain, and vomiting followed by loss of consciousness and routine investigations for common cause were negative; however, there was dramatic response to ASV. Thus, we wish to emphasize the fact that right from primary care a differential diagnosis of snake bite must be kept in mind even in the absence of obvious history of snake bite, especially in the patients reporting early morning because some species (e.g. Krait) may cause painless bite or the patient may not be able to notice it in their sleep. A high degree of suspicion is required in nonendemic areas and rapid venom antigen detection tests like ELISA can be helpful in establishing the diagnosis as timely administration of ASV is critical for favourable outcomes.

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Conflicts of interest

There are no conflicts of interest.

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