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A Case of Neurotoxic Snake Bite in a Metropolitan City (New Delhi): Pain Abdomen with Anaphylaxis as a Presentation

Shruti Virmani, Avanish Kumar Yadav

From, the Department of Paediatrics, ESI PGIMSR, Basaidarapur, New Delhi, India.

Correspondence to: Dr Avanish Kumar Yadav, Senior medical officer, Department of Paediatrics, ESI PGIMSR, Basaidarapur, New Delhi. Email: dryadavavanish@gmail.com

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ABSTRACT

Snake bites present as life threatening emergencies and are seen more commonly in rural tropical countries. Snake bites have neurotoxic, hemotoxic, myotoxic or mixed presentation. We present a case of neurotoxic snake bite in an urban setting of New Delhi, a metropolitan city, where snake bites aren't common. The patient presented as an acute emergency in casualty, without any previous forthcoming history of snake bite, with a history of pain abdomen, vomiting and signs of anaphylaxis. Rapidly progressive neurological deficit started with ptosis followed by stridor, dyspnoea, respiratory arrest and shock. The patient was given standard management of anaphylaxis and snakebite (polyvalent anti-snake venom and neostigmine) based on WHO guidelines of snake bite. Patient recovered without any neurological deficit within a week. Although snakebites are rare in urban setting, they are reported. Neurotoxic snakebites can occur in sleep by kraits and can present as catastrophic life threatening emergency in early morning hours, as in our case.

Keywords: Snake Bite, Envenomation, Anaphylaxis, Neurotoxicity.

Snake bite presents as a life threatening emergency in rural areas of countries like India with an incidence of 4.3 per 100,000 populations and mortality of 20% [1]. The 4 major medically important species of venomous snakes are Naja naja (cobra), Bungarus caeruleus (Krait), Daboia russelii (Russell's viper), Echis carinatum (saw-scaled viper) [2]. Snakes were formerly classified as neurotoxic (cobra, kraits), haemotoxic (viper) and myotoxic (sea snakes); however, it is a well established fact that every species can produce myriads of manifestations [3].

In an Indian study, Saravu K et al showed that hemotoxic and neurotoxic envenomations were observed in 73.68% and 19.73% of cases respectively while hemo-neurotoxic manifestations were seen in 5.26% of cases [4]. Rarely, neurotoxic snake bite can also present with anaphylaxis [2]. Snake bites can happen at home by cobra which dwells on roof tops/under the floors and by Kraits which nocturnaly enter homes in search of prey like mice or lizard. Neurotoxic snake bites especially krait can present with painless bite without local inflammatory signs [1, 2].

CASE REPORT

An eleven year old girl brought to the pediatric casualty with the history of pain in abdomen and vomiting in the early morning. Pain abdomen was of moderate intensity, diffuse, not associated with fever, diarrhea or constipation. She received some symptomatic treatment outside and was referred to pediatrics casualty. The child walked into paediatic emergency room (ER) supported by her parents. She was conscious but dysphonic and had difficulty in looking up. Within minutes, she developed stridor with increasing respiratory distress, pooling of secretions, and

became drowsy with shallow respirations and later, became comatose. Her oxygen saturation fell from 92% (at room air) at presentation to 65% on high flow oxygen and patient had to be intubated and put on intermittent positive pressure ventilation (IPPV) with bag and endotracheal tube. She rapidly developed descending neuromuscular paralysis and lost her own respiratory efforts within 5 min of intubation. At presentation, Glasgow Coma Score (GCS) score was 14 and at GCS score of 8, patient was intubated due to rapidly deteriorating condition. There was no history of diarrhea, fever, constipation, headache, wound/ dental abscess, drug intake, ingestion of preserved foods, poisoning, contacts with pets or any psychological stress at school or home.

On examination at the time admission, she was conscious, afebrile, with pulse rate of 126/min, respiratory rate of 48/min, blood pressure of 120/80 mm-Hg and oxygen saturation of 92% at room air. On general physical examination, there was no pallor/icterus, cyanosis, clubbing, lymphadenopathy or pedal edema. Head and spine were normal. On respiratory system examination, patient was tachypnoeic, bilateral air entry was present with conducted sounds. However, patient soon developed stridor with respiratory distress and oxygen saturation (SPO2) fell. On intubation, there was severe glottic edema with surrounding pooling of hemorrhagic secretions and hyperemia. On abdominal examination, two purpuric spots lying closely with each other (?fang marks) were noted on the lower right side of abdomen. There was no edema, swelling or tenderness at this site. Rest of the skin examination didn't show any rash/petechiae or purpuic spots. On palpation, abdomen was soft with no organomegaly.

On central nervous system examination, patient was initially conscious, oriented, and responding to verbal commands by gestures (as was dysphonic). Ptosis, bilateral mydriasis, and ophthalmoplegia were present and Doll's eye phenomenon was absent. Her power was 4/5 (initially walked with support) then progressed to 0/5 and tone decreased in all 4 limbs, with absent deep tendon reflexes and flexor plantar response.

In absence of any significant history and presence of features suggestive of anaphylaxis with shock and hemorrhagic secretions with glottic edema, patients airway was secured, injection (inj) adrenaline was given intramuscularly along with normal saline boluses and 100% O2 followed by dopamine infusion. As patient did not improve, adrenaline infusion was also started. In view of above clinical findings and rapidly progressive descending neuromuscular paralysis, patient was also given 10 vials of polyvalent anti-snake venom along with the broad spectrum antibiotics, inj. vitamin K and other supportive treatment. Gastric lavage was done and sample was preserved to rule out any poisoning. When patient did not show any response to treatment, patient was given repeat dose of 10 vials of anti-snake venom (ASV), inj. acyclovir, and inj. artesunate along with supportive care. Inj. neostigmine and inj. atropine were also given as per WHO guidelines for snake bite.

On investigation, complete blood counts, bleeding time/clotting time, prothrombin time/activated partial thromboplastin time, international normalized ratio, C-reactive protein, random blood sugar, platelets counts, liver function test, kidney function test with serum electrolytes, electrocardiography, X-ray chest, echocardiography, blood culture were all normal. The initial arterial blood gases (ABG) showed metabolic acidosis with hypoxemia with lactatemia which normalized in repeat ABG after 6 hours. MRI Brain showed a tiny calcified granuloma rest findings were absolutely normal.

Patient showed dramatic response to treatment by showing respiratory efforts within 6 hrs followed by tapering of ventilator settings. On day 2 of admission, patient became conscious and started indicating with finger and communicating with gestures, ventilator settings were further weaned off and vasopressor were tapered off rapidly. On Day3, patient extubated (55 hours after putting on ventilator) and put on CPAP and later on same day, under O2 by mask. All examinations including central nervous system were normal. Child was discharged on day 6 of admission. On follow up after 1 week of discharge, she had no abnormal systemic examination with no neurological deficit and normal higher mental functions. She could also give the history of being bitten by something on abdomen in previous night on the day of presentation.

DISCUSSION

Studies have shown that the common neurological manifestations are ptosis (85.7%) followed by ophthalmoplegia (75%), limb weakness (26.8%),

respiratory (17.9%) and palatal weakness (10.7%), neck muscle weakness (7.1%), and delayed sensory neuropathy (1.8%) [5]. Another study showed that the initial manifestations of neurotoxic snakenbite being ptosis with ophthalmoplegia was followed by bulbar palsy and respiratory muscle weakness. Therefore ptosis and ophthalmoplegia was found to be most common presentation and the earliest sign to appear [6].

Clinical findings in neurotoxic bites in South-East Asian regions were found to be drowsiness, paraesthesiae, taste and smell abnormalities, drooping of eyelids (Ptosis), external ophthalmoplegia, facial muscles paralysis and cranial nerve innervated muscle paralysis, nasal twang of voice or aphonia, regurgitation through nose, dysphagia, respiratory and generalized flaccid paralysis [2]. Ptosis was the most common first sign of neurotoxicity, seen in 100% of patients, followed by external ophthalmoplegia (seen in 94.2%). Other features included dysphagia, dysphonia, broken neck sign etc [1].

Neurological weakness usually appears within hours. The interval between bite and neurological weakness may vary from 1 hour to 10 hours [1,5,6]. In our case, the time interval between bite and presentation of neuromuscular paralysis is also seems to be lesser than 12 hrs. Our patient's prominent initial symptom was pain abdomen with vomiting.Pain abdomen was also reported as a prominent symptom in a study by Kohli et al [7]. Our patient also showed a relatively rarer manifestation of anaphylaxis as reported by Warrel DA et al [2].

Recovery from neurological weakness ranges from hours to weeks. A study showed that recovery from neuromuscular paralysis started within few hours to several days, after administration of antivenom. The duration of complete recovery ranged from few hours to two week [5]. Time of complete recovery of patients, in study by Kohli U et al, was 30 hours after administration of high dose ASV [7]. While in study Robed Amin A et al [1], time lapse between administration of anti-venom and recovery from neurological manifestation ranged from 0.5 to 48 hours (median - 4 hours). In our case this period was found to be 6 to 55 hours. In a study by Robed Amin A et al [1] complete recovery was seen in 34 out of 35 patients of neurotoxic snakebite (97% of cases), only one patient was found to have persistent neurological sequelae. Similarly in a study by Bhattacharya P et al, 12 out of 13 patients of severe neuroparalytic envenomation, recovered completely without any neurological sequlae [6].

In a study, out of 35 patients 3 patients were unconscious at presentation while 2 patients were apneic with imperceptible pulse rate and blood pressure, while rests of the patients were conscious even when they were on ventilator [2]. Our patient retained consciousness till she desaturated. Diagnosis of snake bite can be done by 1) reliable history of being bitten by a snake 2) examination of dead snake brought along the patient 3) ELISA test on venom antigen (wound, serum or urine) being used in some nations [8]. Examination of local wound can also give important clue as in a study, out of 35 neurotoxic snake bites 20 (57%) patients were having typical double fang marks while 13 (37%) showed single bite mark [1]. However in most of the cases, these may not be available and diagnosis of snakebite, especially neurotoxic bite by kraits, depends on clinical presentation.

Studies have shown that we may not find bite marks on exposed part of skin and in that case other unusual sites like scalp/ other hairy areas should be examined after shaving it off [9]. A close mimicker of snakebite is botulism. 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 a differentiating feature from krait bite [10]. However in our case differentiating features were absence of prodrome, wound or abscess including dental abscess, history of contaminated food, rapidly progressive descending paralysis, pooling of secretions, loss of deep tendon reflexes, evidence of marks on abdomen which we suspected could be fang marks and on basis of this and as per WHO criteria (vide infra) we decided to go ahead with use of polyvalent anti-snake venom despite an urban setting and absence of any history of snakebite.

Polyvalent anti-snake venom is the cornerstone of the management of snakebites. As per WHO guidelines [2], anti-venom treatment is recommended if a patient with proven or suspected snakebite has developed one or more of the following signs: 1) Haemostatic abnormalities: spontaneous systemic bleeding (clinical), coagulopathy (20 whole blood clotting test or other laboratory tests such as prothrombin time) or thrombocytopenia (<100,000 /mm^3) (laboratory) 2) Neurotoxic signs: ptosis, external ophthalmoplegis, paralysis etc (clinical) and 3)

Cardiovascular abnormalities: hypotension, shock, cardiac arrhythmia(clinical), and abnormal ECG.

In our case, patient resided in relatively hilly and bushy area of urban Delhi and presented initially with pain abdomen, vomiting, features of anaphylaxis along with ptosis, mydriasis, ophthalmoplegia and rapidly progressive descending type neuromuscular paralysis requiring intubation. In view of suspected snake bite, she was given polyvalent anti-snake venom, mechanical ventilation along with other supportive care. Recovery was noted within 6 hrs in the form of spontaneous respiratory efforts and showed complete recovery on 3rd day of admission and was discharged on day 6 with complete neurological examination being normal at 3 months and 6 months post event.

CONCLUSION

We report this case because snake bites in a metropolitan city like New Delhi are rare and high degree of suspicion is required to prevent mortality and morbidity. We emphasize that cases presenting with ptosis and rapidly progressive descending type neuromuscular paralysis in the background of other features (pain abdomen, anaphylaxis in this case), neurotoxic snake bite should be kept as one of the possibility. Snake bite such as krait bite (most probably in this case) can be painless and without any local inflammatory sign.

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