

Case Report

Delayed neurological manifestation in viper bite despite anti-snake venom therapy

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ABSTRACT

Envenomation due to snake bite is an acute life threatening medical emergency. Among the different families of snakes, viper bites are known to cause local manifestations like cellulitis, blebs, compartment syndrome; as well as systemic manifestations which include neurological, hematological and renal failure. This is a case of a middle aged man who presented with viper bite. He was given anti-snake venom (ASV) and became asymptomatic. After about 72 hours of ASV therapy, he started developing generalised paralysis. He was given another course of ASV, following which he recovered completely. To the best of knowledge, this form of delayed neurological manifestations following viper bite, despite receiving ASV has not been reported yet.

Keywords: Anti-snake venom, Neurological manifestations, Snake bite

INTRODUCTION

About 215 species of snakes have been identified in India. Of these, around 52 are poisonous. All these species of snakes have been classified under 3 major families, i.e. Elapidae (common cobra, king cobra, krait), Viperidae (russell's viper, saw scaled viper, pit viper) and Hydrophiidae (sea snakes). Elapidae group are mainly neurotoxic, viperidae haematotoxic and hydrophiidae myotoxic. The snake venom is composed of enzymes, polynucleotide toxins, non-toxin proteins, carbohydrates, metals, lipids, free amino acids, nucleotides and biogenic amines. Local manifestations of viper bites include blisters at bite site, local swelling and painful lymphadenopathy. Haemostatic abnormalities like bleeding from bite site and systemic haemorrhage are seen. Neurologically, visual disturbances, respiratory and generalized paralysis have been described.¹ In this case, the patient presented with right lower limb cellulitis and hypotension following viper bite. He was treated with anti-snake venom (ASV), following which he became

asymptomatic. After about 3 days, he started developing generalised paralysis. Such a delayed neurological presentation, despite ASV therapy, has not been reported earlier. He improved after another course of ASV.

CASE REPORT

A 46 year old male presented to the Emergency department at night with alleged history of snake bite while he was walking along with his friends in the field. His friends killed the snake and identified it as viper. He was brought to our hospital within 1 hour of the bite.

On presentation, he was conscious, oriented and anxious. He had a blood pressure of 90/60 mmHg, heart rate of 130 beats/minute and respiratory rate of 28/minute, with saturation 94% in room air. Fang marks were visible above the right lateral malleolus with no active bleeding (Figure 1). There was mild swelling of the right lower limb extending almost up to the knee, with associated redness and tenderness, with mild local rise in

temperature (Figure 2). He had tender right inguinal lymphadenopathy. His systemic examinations were normal.



Figure 1: Fang marks above the right lateral malleolus.



Figure 2: Right lower limb cellulitis.

His complete blood count showed haemoconcentration with Hb of 16.5 g/dL (13-16), haematocrit of 51, total counts 13,000/ cmm (4000-10,000) with differential as N85 L15 and platelet count of 100,000/cmm (150,000-450,000). His renal functions were deranged with urea 62 mg% (20- 40) and creatinine 1.6 mg% (0.4-1.4); and liver functions showed total bilirubin 2.7 mg% (0.3-1.3), direct bilirubin 1.5 mg% (0.1- 0.4), SGOT 154 IU/ L (12-38), SGPT 80 IU/ L (7- 41), ALP 204 IU/ L (33-96), total protein 5.5 gm% (6.7-8.6), albumin 3.5 gm% (3.5-5.5) and GGT 40 U/ L (9-58). His 20-minute whole blood clotting test (20WBCT) was suggestive of venom-induced consumption coagulopathy (as the blood did not

clot). Serum electrolytes were normal. Urine microscopy showed numerous RBC, but urine myoglobin was negative. ECG and chest X-ray were normal.

He was given 100 ml (10 vials) of ASV in 200 ml of normal saline intravenously over 1 hour. He did not develop any anaphylactic reaction to ASV. Injection cloxacillin was started for cellulitis. Normal saline and pantoprazole injections were also given. 20 WBCT was repeated 6 hours after ASV administration and was normal. Hence, no further doses of ASV were given. The next day, his vitals started stabilizing. His lower limb cellulitis also decreased. His renal and liver functions started normalizing. Complete blood count showed Hb of 15 g/dL with haematocrit of 46, total counts 12,100/ cmm and platelets 120,000/ cmm. There were no bleeding manifestations.

On day 3 of admission, he started developing flaccid paralysis of upper and lower limbs. His lower limb power was 1/5 bilaterally with mute plantars and upper limb power 3/5 bilaterally. His deep tendon reflexes were diminished with no sensory involvement. Neck power was preserved. Cranial nerves were normal. He was conscious and oriented with no ptosis or respiratory distress. Emergency CT brain and whole spine were taken which turned out to be normal. He was given another course of 100 ml ASV in 200 ml normal saline over 1 hour. In less than 1 hour following ASV administration, his lower limb power started improving and in about 3 hours he was completely asymptomatic. His next 4 days in hospital were uneventful; and was discharged on day 7 of admission after complete normalisation of vitals, cellulitis and blood reports. An informed consent was obtained from the patient to publish this paper.

DISCUSSION

Snake bites are considered as medical emergencies, affecting mainly the rural population. The incidence is particularly higher in South East Asian countries. The snake venom is not a single toxin. It comprises of proteins which include enzymes, non-enzymatic polypeptide toxins and non-toxic proteins. The non-protein components are carbohydrates, lipids, metals and biogenic amines. Enzymes like phospholipase A₂ damage mitochondria, RBC, WBC, platelets, peripheral nerve endings, skeletal muscles and vascular endothelium. Hyaluronidase is responsible for spread of venom through tissues. Proteolytic enzymes cause local changes like blisters, edema and bruising. The neurotoxins are both presynaptic and postsynaptic. The presynaptic phospholipase A₂ (β neurotoxins) damage nerve endings at the neuromuscular junctions, resulting in failure of acetylcholine release and paralysis. The postsynaptic neurotoxins (α neurotoxins) bind to the $\alpha 1$ nicotinic acetylcholine receptors at the motor end plates of skeletal muscles causing generalized flaccid paralysis. Death can occur due to bulbar and respiratory paralysis.²

Viper bites can cause local envenomation. Swelling may appear immediately, which can spread involving the whole limb and adjacent trunk. This is associated with tender regional lymphadenopathy. There may be persistent bleeding from the fang marks. Bruising along the path of superficial lymphatics is common. Bites in areas draining into tight fascial compartments can cause compartment syndrome. Deep vein thrombosis is rare. Haemostatic abnormalities include spontaneous systemic haemorrhage mainly in the gingival sulci, hematuria, intracranial, subconjunctival and gastrointestinal haemorrhage. Generalised or respiratory paralysis, ptosis, diplopia, dysphagia etc are the common effects of neurotoxicity. Other features include intravascular haemolysis, circulatory shock and renal failure.²

The indications for ASV treatment as outlined by the WHO are:

Systemic envenoming

Haemostatic abnormalities

Spontaneous systemic bleeding (clinical), coagulopathy (20WBCT or other laboratory tests such as prothrombin time) or thrombocytopenia (<100 x 10⁹/litre or 100 000/cu mm) (laboratory).

Neurotoxic signs

Ptosis, external ophthalmoplegia, paralysis etc (clinical).

Cardiovascular abnormalities

Hypotension, shock, cardiac arrhythmia (clinical), abnormal ECG.

Acute kidney injury (renal failure)

Oliguria/anuria (clinical), rising blood creatinine/ urea (laboratory)

Haemoglobin-/myoglobinuria, dark brown urine (clinical), urine dipsticks, other evidence of intravascular haemolysis or generalised rhabdomyolysis (muscle aches and pains, hyperkalaemia) (clinical, laboratory). Supporting laboratory evidence of systemic envenoming.

Local envenoming

- Local swelling involving more than half of the bitten limb (in the absence of a tourniquet) within 48 hours of the bite. Swelling after bites on the digits (toes and especially fingers)
- Rapid extension of swelling (for example, beyond the wrist or ankle within a few hours of bite on the hands or feet)
- Development of an enlarged tender lymph node draining the bitten limb.³

The ASV available in India is polyvalent and is effective against all the four common species (Russell's viper, common cobra, common Krait and saw-scaled viper). Prophylactic medication to prevent ASV reactions may not be required. If so then the regimens normally recommended are hydrocortisone (100 mg) + antihistamine or 0.25-0.3 mg adrenaline subcutaneously.⁴ 8-10 vials of ASV are given for envenomation; where each vial is 10 ml of reconstituted ASV. Further doses will depend on the response to the initial dose. The 20 WBCT should be repeated only after 6 hours of ASV administration due to the inability of the liver to replace clotting factors in less than 6 hours. If WBCT is more than 20 minutes, then 5-10 vials of ASV is administered again. Local administration of ASV near or at the bite site should not be done as it is ineffective and can predispose to compartment syndrome.¹ Early anaphylaxis, pyrogenic reaction (due to ASV contamination by endotoxin like compounds) and late serum sickness are the commonly seen reactions to ASV therapy.²

Neurological symptoms following snake bite are commonly experienced within 6 hours. The symptoms include ptosis (85.7%), ophthalmoplegia (75%), limb weakness (26.8%), respiratory failure (17.9%), palatal weakness (10.7%) and neck muscle weakness (7.1%).⁵ Encephalopathy is a rare presentation.⁶ Recovery is usually seen within a few hours to several days of ASV administration.⁷

CONCLUSION

Neurological deficits may be seen with viper bites. However, this must be the first case in which the patient developed delayed neurotoxicity in the form of flaccid paralysis, despite receiving ASV. Hence, following snake bite the patients should be monitored for at least 1 week in view of delayed neurological effects and late serum sickness associated with ASV administration.

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