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Short Article

Neurotoxic Presentation of Russell's Viper bite case: A report from North India

Srishti Sharma[1], Kriti Sharma[2], Omesh Bharti[3]

[1]Regional Hospital, Una, Himachal Pradesh, India
[2]Ex. Medical Officer, DDU Hospital, Shimla, Himachal Pradesh, India
[3]Dy. Director Health Services cum State Epidemiologist, Himachal Pradesh, India
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I N F O

Corresponding Author:
Omesh Bharti, Dy. Director Health Services cum State Epidemiologist, Himachal Pradesh, India
E-mail Id:
bhartiomesh@yahoo.com
Orcid Id:
https://orcid.org/0000-0001-5178-1503
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A B S T R A C T

Russell's Viper (*Daboia russelii*) is a highly venomous snake species prevalent in Southeast Asia. Russell's viper is one of the "BIG FOUR" snakes responsible for most venomous bites and deaths in India. Envenomation in Russell's viper bite is commonly associated with coagulopathy, renal dysfunction and local tissue damage. While haematological and nephrotoxic complications are well documented in Russell's viper bites, neurological sequelae as first presentations are rarely seen and reported. Neurological symptoms in Russell's viper bites, such as ptosis, ophthalmoplegia and respiratory paralysis, can mimic elapid envenomation and demand prompt identification and Anti Snake Venom (ASV) administration. We report a case of successful management of a patient presenting with neurotoxic features following a Russell's viper bite with a focus on early recognition, timely administration of ASV and supportive care. This is probably the first report from North India of a Russell's viper bite case presenting as neuropathy and is of high academic interest not only for clinicians but also for venom analysis to find the neurotoxins responsible.

Keywords: Russell's Viper, Neuropathy, Paralysis, Atropine, Neostigmine, Ca. Gluconate

Introduction

India is estimated to have the highest snakebite mortality in the world. World Health Organisation (WHO) estimates place the number of bites and deaths due to snakebite as variable.[1] Recent national estimates predict snakebite numbers to be 129,325 per annum with 5039 deaths in India.[2] A recent study has identified more than 20 known species that are venomous, and of these four- namely the common cobra (Naja naja), Russell's viper (*Daboia russelii*), the saw- scaled viper (Echis carinatus) and the common krait (Bungarus caeruleus) are highly venomous and believed to be responsible for most of the deaths in India,[3] for which Indian ASV is used for treatment. Bleeding and clotting disorders are caused by Viperidae, like spontaneous systemic bleeding from gums, epistaxis, intracranial haemorrhage, haemoptysis, haematemesis, rectal bleeding or melena, bleeding into the mucosae skin (petechiae, purpura, ecchymosis) and retina. Although Russell's viper venom may contain neurotoxins as well, clinical manifestation of neurotoxicity as a presenting sign is rare.[4,5] A Sri Lankan study demonstrates that neurotoxicity following Sri Lankan Russell's viper envenoming is primarily due to the pre-synaptic neurotoxin U1-viperitoxin-Dr1a and, the Indian polyvalent antivenom, at the recommended concentration, only partially prevented the neurotoxic

effects of U1-viperitoxin-Dr1a.[6] Neurological symptoms in Russell's viper bites, such as ptosis, ophthalmoplegia and respiratory paralysis, can mimic elapid envenomation and demand prompt identification and Anti Snake Venom (ASV) administration. We report a case of successful management of a patient presenting with neurotoxic features following a Russell's viper bite, with a focus on early recognition, timely administration of ASV and supportive care.

Case Presentation

A 34-year-old female was brought to the emergency department within 15 minutes of a suspected snake bite on the little finger of her left hand, as two fang marks were present, sustained while working in a field in the rural region of District Una, Himachal Pradesh. The snake was identified as Russell's viper in the photograph they showed to the author on their mobile phone. As per the herpetologist's viewpoint (Mr Vishal Santra), the snake appears to be in a ready attacking position (Fig. 1). The herpetologist was of the view that the snake has already eaten some big prey and is in an aggressive posture.

Figure 1.Russell's Viper in ready attacking position after bite

Initial Presentation

- **Vital Signs:** Pulse 84/min, BP 137/72, SpO_2 was 98% on room air, RR 20/min, Temp. 97 F
- **Local signs:** Fang marks present on the little finger of the left hand.
- **Systemic Signs:** No bleeding or haematuria, numbness in the left arm present; however, other vitals were stable. Within 10 minutes, the patient developed:

Muscle Weakness in the left arm followed by complete left arm paralysis and not being able to lift the left arm. Bilateral drooping of eyelids and sluggish eye movements and shortness of breath with difficulty in breathing.

Investigation

Whole Blood Clotting Test (WBCT): less than 20 mins (15 mins)

Hb 10.4

TLC 7960

Platelets 221

Blood group B+

Random Blood Sugar: 119

Serum Urea 25

Serum Creatinine 0.70

Total Bilirubin 0.50

SGOT: 21

SGPT: 35

ALP: 52

HIV: NR

HCV:NR

Diagnosis

Based on the snake identification and presenting symptoms, a diagnosis of neurotoxic envenomation due to Russell's viper was made. Before ASV administration, high- risk consent was taken. Later, consent to publish was also taken.

Management

The IV line was secured, and IV fluid normal saline (NS) was started.

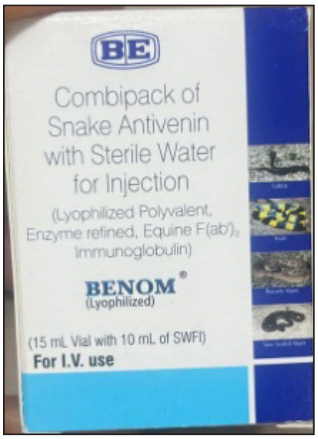
Inj. TT 0.5mg i/m stat

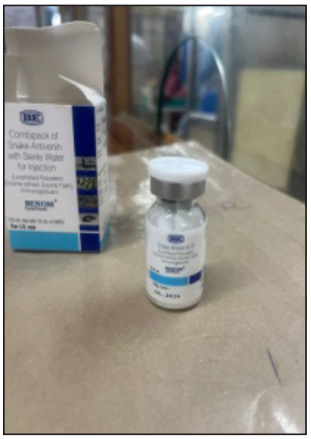
Inj. Avil 2 ml i/v stat

Inj. Hydrocortisone 100mg i/v stat

O_2 inhalation at 6-8 L/min intubation equipment kept ready if SpO_2 < 90%)

10 vials of ASV in 500 ml NS over 1 hour after the test dose.





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(Batch no. A4002624, Mfg. date 07/24, Exp.date 06/2028 Manufactured by Biological E Ltd)

Inj. Calcium Gluconate 1 vial, 10 ml (950 mg) in Dextrose 5% over 20 mins.Inj. Atropine 0.6mg IV stat

Inj. Neostigmine 0.5mg IV stat

I/O charting of fluids given IV.

Monitoring of neurological signs, respiratory rate, oxygen saturation, and repeat WBCT every 6 hours. The patient completely recovered and was discharged the next day with all vitals restored to normal.

Discussion

Venoms of Viperidae contain serine proteases and other procoagulant enzymes that are thrombin-like or activate factor X, prothrombin and other clotting factors. These enzymes stimulate blood clotting with the formation of fibrin in the bloodstream. Paradoxically, this process results incoaguable blood because most of the fibrin clot is broken down immediately by the body's own plasmin fibrinolytic system, and sometimes within 30 minutes of the bite, the levels of clotting factors are depleted (consumption coagulopathy). Some species of vipers (Mojave/Russell's viper) have postsynaptic (α) neurotoxins such as α-bungarotoxin and cobrotoxin, which consist of 60-62 or 66-74 amino acids. They bind to acetylcholine receptors at the motor endplate. Presynaptic (β) neurotoxins, such as β- bungarotoxin, crotoxin, and taipoxin, contain 120-140 amino acids and a phospholipase A subunit. These release acetylcholine at the nerve endings at neuromuscular junctions and then damage the endings, preventing further release of transmitter and producing neurotoxicity.[7] In Sri Lanka, the failure of Indian (Haffkine) antivenom showed that 23 patients with systemic envenomation after proven bites had swelling at the bite site (73 per cent), neurotoxicity in the form of external ophthalmoplegia (82 per cent) and 77 per cent had ptosis. Incoagulable blood was found in 59 per cent, but only 36 per cent had spontaneous bleeding. Laboratory studies showed evidence of a severe clotting disorder: fibrinogen was often depleted, as were factors V and X.[8] Despite the documented low efficacy of ASV in North India RV venoms.,[9] our patient responded to 10 vials of Indian polyvalent ASV; this had some relation with the early ASV administration as soon as the symptom of neuropathy started. Viper bite is associated with coagulation abnormalities and renal failure, with occasional reports of neurotoxicity, pituitary necrosis and increased vascular permeability. Considerable geographical variation in clinical presentation has been described following bites by some species of snakes, including Russell's vipers, whereas treatment with potent specific antivenom rapidly controls bleeding and clotting disorders but may not reverse nephrotoxicity and shock.[10] Neurological manifestations of Russell's viper bite, although previously rare, are being increasingly recognised. The neurotoxins in the venom may cause:

- Presynaptic blockade (impairing acetylcholine release)
- Cranial nerve involvement
- Bulbar palsy, mimicking elapid bite

Producers of antivenom must utilise an understanding of such variability in selecting sources of venom for antivenom production to ensure representation of all venom types required within each antivenom.[11]

Differentiation from cobra/krait envenomation is crucial for epidemiology and prognosis but may not change immediate management due to the use of Indian polyvalent ASV. Early administration of ASV is the only specific treatment. Supportive respiratory care can be life- saving, especially in rural settings where ventilators may not be available. Injection of calcium gluconate may have a role in the reversal of pre-synaptic neuromuscular blockade; however, we also gave an injection of atropine-neostigmine to ameliorate any post-synaptic effect of venom that may be there or if there is mistaken snake identity of the patient, as sometimes a different snake is pictured than the actual one that may have bitten the patient. Atropine-neostigmine was also used in Russell's viper bite in other settings also for reversal of neurotoxicity.[12] Improvement by atropine and neostigmine indicates a cobra bite. A few Nilgiri Russell's viper bite victims also improve with this regimen.[13]

Conclusion

This is probably the first report from North India of a Russell's viper bite case presenting as neuropathy and is of high academic interest not only for clinicians but also for venom analysis to find neurotoxins responsible.

This case highlights the importance of:

- Early identification of neurological signs in Russell's viper bite
- Prompt ASV administration
- Inj. Calcium Gluconate 1 vial, 10 ml (950 mg) in Dextrose 5% over 20 mins
- Inj. Atropine 0.6mg IV stat
- Inj. Neostigmine 0.5mg IV stat
- Multidisciplinary supportive care

Neurotoxic symptoms in viper bites must not be underestimated. Timely intervention can significantly reduce morbidity and mortality in such cases.

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