

# Viper bite causing an isolated lower motor neuron-type of facial palsy

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## ABSTRACT

We describe an unusual case of viper (*Daboia russelii*) bite in a 48-year-old man from the state of Karnataka in southern India. He presented in a hypotensive state with a left lower motor neuron-type of facial palsy, necrosis at the site of the bite and acute renal failure. His laboratory parameters revealed renal failure and deranged coagulation parameters. He was treated with intravenous antibiotics and polyvalent antiserum venom, and dialysed in view of the renal failure. His renal function and coagulation abnormalities improved, and the facial palsy recovered with the treatment. The snake bite located away from the face, the facial palsy occurring a few hours after the venom injection and the rapid recovery following antivenin administration, support that the palsy was a direct result of systemic envenomation. To the best of our knowledge, an isolated lower motor neuron-type of facial palsy as a manifestation of systemic toxicity of a viper bite, has not been previously reported.

**Keywords:** anti-snake venom, facial palsy, renal failure, snake envenomation, viper bite

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## INTRODUCTION

The Russell's viper, one of the most dangerous snakes in Asia along with cobras and kraits, contributes to the snake bite mortality and morbidity each year. It is a member of the five species of snakes which are responsible for most Indian snakebite mortalities, and are thus of medical importance in India.<sup>(1)</sup> Viper envenomation causes local manifestations such as pain, oedema, bleeding and necrosis. This may be accompanied by systemic features like hypotension or shock, acute renal failure, and bleeding and blood clotting abnormalities. The other clinical manifestations include weakness, dizziness and nausea. Neurological manifestations due to viper envenomation have also been very well described.<sup>(2)</sup>

## CASE REPORT

A 48-year-old man, a farmer by occupation, was



**Fig. 1** Photograph shows necrosis of the dorsum of the right foot following a viper bite, with exposure of the underlying tendons. The limb also shows cellulitis and blister formation.



**Fig. 2** Photograph shows a partially-recovered left lower motor neuron-type of facial palsy with a deviation of the angle of the mouth to the right on Day 2 of presentation.

bitten by a viper (*Daboia russelii*; the dead snake was identified by the experts from the forensic department) on the dorsum of his right foot, in the evening prior to his presentation, while he was working in the field. This was soon followed by continuous bleeding from the site, with swelling, redness and severe pain. After three hours, he had a few episodes of vomiting and noticed a deviation of the angle of his mouth to the right side and an inability to close his left eye. There was no history of other neurological deficits. The next morning, he noticed a decrease in urine output, and that the leg swelling had been progressively increasing.

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**Table I. List of WHO classification of snakes of medical significance in India.<sup>(1)</sup>**

Class	Morbidity/mortality	Type of snake
1	Commonly cause death or serious disability.	cobra, Russell's viper, saw-scaled viper
2	Uncommonly cause bites but are recorded to cause serious effects (death or local necrosis).	krait, King Cobra
3	Commonly cause bites but serious effects are very uncommon.	White-lipped pitviper

The patient presented to us 24 hours after the bite. At presentation, he was pale, hypotensive (blood pressure [BP] 90/60 mmHg) and tachycardiac (heart rate 110/min). The dorsal aspect of the right foot was swollen and necrotic, and cellulitis of the limb was also noticed (Fig. 1). The neurological examination revealed a left lower motor neuron-type of facial palsy, with a loss of the nasolabial fold and weakness in the eye closure on the left side, as well as a deviation of the angle of the mouth to the right. Bell's phenomenon was noticed in the left eye. No other neurological deficit was noticed.

His initial laboratory parameters revealed a haemoglobin level of 9.8 g/dL, leucocytosis 25,000/mm<sup>3</sup>, thrombocytopenia 40,000/mm<sup>3</sup>, and serum creatinine 12.5 mg/dL (1,105 µmol/L). There was a derangement of coagulation parameters (bleeding time 3 minutes; clotting time 6 minutes; prothrombin time 20 s [vs. control 13.5 s]; activated partial thromboplastin time 31 s [vs. control 28.2 s]) and the serum creatine kinase was 1,608 mg/dL. Liver function test, chest radiograph and electrocardiograph were all normal. The patient was referred to the neurology unit, and computed tomography of the brain was done, but it did not reveal any bleeding or infarct in the brain.

The patient was started on intravenous antibiotics for the local wound infection, intravenous fluids (with which the BP normalised) and haemodialysis. In view of the signs of local and systemic envenomation, polyvalent antiserum venom (antivenin against cobra, common krait, Russell's viper and saw-scaled viper) was administered in the intensive care unit on the day of admission. Within six hours, the coagulation parameters improved and the facial palsy started to recover rapidly (Fig. 2). A total of 12 vials were required over a period of 24 hours to completely normalise the coagulation abnormalities, and the facial palsy also recovered completely. Tetanus immunisation was given after the coagulation abnormalities were corrected. His renal parameters improved after ten sessions of haemodialysis (over a period of two weeks), and his haemoglobin levels also improved. Wound debridement and daily dressings were done. Split skin grafting was applied once the granulation tissue appeared.

## DISCUSSION

Previously, four species of snakes were recognised in India: The Indian cobra (*Naja naja*), the common krait (*Bungarus caeruleus*), the Russell's viper (*Daboia russelii*) and the saw-scaled viper (*Echis carinatus*). In 1981, the World Health Organization proposed a different classification for the identification of snakes of medical significance, as the previous concept of the "big four" failed to include all the currently known snakes of medical significance in India (Table I). The hump-nosed pitviper (*Hypnale hypnale*), previously considered harmless, should also be added to class 2 in the list.<sup>(1)</sup> Viper envenomation causes a local inflammatory syndrome with clinical manifestations such as pain and oedema. This may be accompanied by systemic features like hypotension or shock. Some of the common clinical manifestations include pain, weakness, dizziness, nausea and thrombocytopenia. Envenomation may be complicated by haemorrhagic syndrome and necrotic complications around the bite, requiring emergency medical attention and surgical intervention.<sup>(3)</sup>

Neurological complications following viper bites have been very well described in the literature.<sup>(2)</sup> Neurotoxicity from the Sri Lankan and southern Indian *Daboia russelii* is well recognised.<sup>(4,5)</sup> Frequently-reported neurological deficits include cranial nerve palsy manifesting as ptosis, ophthalmoplegia, dysphonia, dysphagia, drooling and diplopia, with more severe presentations leading to respiratory failure and death. The neurological toxicity results from the systemic effects of the neurotoxin and is not related to mass effect, intracranial haemorrhage or infarct and coagulopathy. They block transmission at the neuromuscular junction. Other Asian viper species capable of causing neurotoxicity include the mamushis (*Gloydius* spp.), but these are found mainly in China and Japan. Neurotoxicity has also been described in other viper species.

A description of isolated lower facial nerve palsy caused by a *Daboia russelii* bite has not been specifically described in previous case reports, although facial paralysis is a recognised complication. Other examples of the neurological deficits following viper

bites reported from different parts of the world include acute descending paralysis,<sup>(2)</sup> multiple neurological deficits with haemorrhagic infarcts in the brain,<sup>(6)</sup> acute paraplegia,<sup>(7)</sup> brainstem infarction<sup>(8)</sup> and aphasia.<sup>(9)</sup> Most of these manifestations were attributed to coagulopathy. Only one case of isolated facial palsy has been reported in the literature, where the paresis of the left facial frontalis muscle was hypothesised to be due to the unusual location of the viper bite in the left frontotemporal area. The explanation given in that particular case was the possible exposure of the muscle to the venom, spreading directly from the injection site and destroying the nerve terminals in the muscle tissue; this was supported by the electromyographic test results.<sup>(10)</sup>

In our case, as the bite was away from the face and over the dorsum of the right foot, facial palsy occurred a few hours after the venom injection and rapid recovery was observed following antivenin administration. These factors support that it was a consequence of systemic envenomation, and illustrate the varied neurological presentations caused by the viper envenomation. Complete recovery is one of the well-recognised features of snakebites. However, cases with permanent damage can occur, especially in venom-induced cerebrovascular accidents. Recovery can be rapid, occurring within hours, or it may be delayed for weeks.

The hospital management consists of a rapid thorough physical examination and close monitoring, fluid resuscitation with normal saline in cases of shock and vasopressors if there is no response to fluid resuscitation. Invasive haemodynamic monitoring with central venous pressure monitoring is needed in cases with persistent shock.<sup>(11)</sup> The antivenin therapy is indicated when there is clinical or laboratory evidence

of envenomation. Because of the risk of anaphylactic reactions or serum sickness, antivenin should be given with great caution.<sup>(12)</sup> Whether or not antivenin is given, any patient with signs of venom poisoning should be observed in hospital for 24 hours. Tetanus prophylaxis is advised in all cases.

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