

## Snake bite: An unusual cause of ischaemic stroke

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### CASE STUDY

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### ABSTRACT

Snake bite continues to be one of the major and potential contributors for morbidity and mortality in India. Intracranial haemorrhage causing neurological deficits is a known entity due to snake bite but presentation with infarction is rare. The authors report a case of a healthy middle aged female who presented within hours of envenomation with altered sensorium and evaluation revealed left hemiplegia with deranged coagulation profile and multiple ischaemic infarcts instead of haemorrhage as a consequence of vasculotoxic envenomation due to viper bite.

#### Key Words

Snake bite, hemiplegia, infarction

#### Implications for Practice:

##### 1. What is known about this subject?

Snake bite presenting as multiple ischaemic infarcts is very uncommon and rarely reported.

##### 2. What new information is offered in this case study?

Correction of coagulation parameters should not be the driving criteria for termination of anti-snake venom therapy.

Instead, overall recovery patterns should be considered for treatment and management.

##### 3. What are the implications for research, policy, or practice?

Continuation of anti-snake venom despite normalisation of the haematological parameters may prevent potential vasculotoxic effect of the venom and further vital organ damage.

### Background

Snake bite is a common medical emergency in India and a predominant cause of fatality and morbidity. Identifying the species of snake is of prime importance to administer appropriate treatment and save resources.<sup>1</sup> The snake species responsible for most envenomation in India are the great four-namely Russell's viper (*Dabiola russelii*), saw-scaled viper (*Echis carinatus*), common cobra (*Naja naja*), and common krait (*Bungarus caeruleus*).<sup>2</sup> The presentation of the patients with bites due to different species usually varies and mostly obvious from the history. Vasculotoxicity is a pathognomonic feature of a viper bite, which leads to a wide spectrum of various neurological complications, which usually manifest as intra-cerebral or subarachnoid haemorrhage, often with focal or generalised manifestations.<sup>3,4</sup> However, snake bite being the cause for infarcts in specific vascular territories is a very rare entity and has been reported in very few cases.<sup>5</sup>

The authors report a case of an adult female with no known pre-morbidities presenting with multiple cerebral infarcts following vasculotoxic envenomation.

### Case details

A 45-year-old adult female with history of snake bite on the right ankle was brought to the emergency department of our institution following referral from a local hospital. She had no significant past medical or surgical illness. History revealed that the patient had profuse bleeding from the bite site, developed laboured breathing and had lost consciousness within three hours on her way to a local

hospital. She was immediately intubated in view of her poor Glasgow coma scale (GCS) score and was administered twenty vials of polyvalent anti-snake venom (ASV) at a local hospital due to deranged coagulation profile and was referred to our hospital. There was specifically no history suggestive of an old cerebro-vascular accident or any cardiac illness in the past.

Examination revealed a comatose, intubated patient, with a Glasgow coma scale (GCS) of E<sub>1</sub>V<sub>7</sub>M<sub>1</sub>. There was active bleeding from the bite site and fang marks were present. There was bleeding from the oral cavity and fresh blood was aspirated from the stomach as well. She had hypotension; pupils were 4mm in size, bilaterally equal, sluggishly reacting to light and the oculocephalic reflex was present. She had obvious paucity of movements on the left side with left extensor plantar response. Other systems were normal.

The haematological profile revealed an initial prolonged whole blood clotting time of 20 minutes (20WBCT). Her haemoglobin (Hb) was 14.7gm/dl, total counts were elevated to 16,000cells/mm<sup>3</sup> and platelets were 153,000cells/mm<sup>3</sup>. Sugars, renal and hepatic parameters were within normal limits. Creatine phosphokinase (CPK) was elevated (2076IU/L) and lactate dehydrogenase (LDH) was 496IU/L. The coagulation parameters, prothrombin time (PT) and INR, activated partial thromboplastin time (aPTT) were prolonged (>120 seconds). Thromboelastography (TEG) assay revealed absence of multiple coagulation factors. Initial chest X-ray, ECG and ECHO were normal. Lower limb venous Doppler was normal. CT brain was done at admission and it was normal. She was restarted on polyvalent ASV in addition to Inotropes and broad spectrum antibiotics. The area around the snake bite was already necrosed at admission so an emergency debridement was done. Fresh frozen plasma (FFP) was also transfused to mitigate the active bleeding. Her platelet counts gradually fell suggesting an active consumptive coagulopathy. Intravenous polyvalent ASV was continued on day 1 (16 vials).

On day 2, the bleeding stopped and coagulation parameters showed an improving trend, however as she had left sided paucity of movements with extensor plantar on the same side. An MRI of the brain revealed acute to sub-acute infarcts in bilateral cerebellar hemispheres, bilateral thalami, bilateral frontal and parietal lobes, right centrum semi-ovale, right temporal lobe and right half of midbrain with haemorrhagic transformation (Figures 1-6).

ASV was continued and she was started on anti-cerebral oedema measures with intravenous mannitol, following which her GCS score gradually improved. Her coagulation profile and platelet count became normal by fourth day and ASV was stopped on day 4. She was successfully extubated on day five of hospitalisation. Supportive measures and physiotherapy was started. Antiplatelets were not preferred in view of haemorrhagic transformation. She was on an improving trend and power on day 14 was 3/5 (Medical research council grade) in the left upper and lower limb.

## Discussion

The commonest cause of snake bite in India is due to viper species. It presents with a wide variety of local complications including gangrene and necrosis of the bite site and also systemic vasculotoxicity. Viper venom contains pro-coagulant enzymes that are thrombin like and activate the coagulation cascade causing consumption of clotting factors and subsequently forming intravascular fibrin thrombi. This causes consumptive coagulopathy and leads to bleeding. The viper venom also contains several anticoagulant proteins that activate factors V, IX, X, XIII or cause fibrinolysis and subsequently haemorrhage.<sup>6</sup> Due to this 'pro-bleeding' milieu, the common neurological manifestations are due to intra-cerebral or subarachnoid haemorrhage, with rare occurrence of ischaemic infarctions. Hence various mechanisms have been proposed for the occurrence of infarctions.<sup>7-9</sup>

The first being, the pro-coagulant effects of the viper venom can cause the formation of intravascular fibrin thrombi, which may cause occlusion of the cerebral vessels, causing infarction.<sup>10</sup> This is a likely cause considering the consumptive coagulopathy that our patient was manifesting. The second mechanism suggested is hypotension causing infarcts in the watershed areas of the brain.<sup>11</sup> Although the patient in the study had hypotension, it was treated immediately with inotropic support to maintain a MAP (mean arterial pressure) of >65mmHg and moreover the distributions of infarcts in the patient were not in the watershed territories.

Finally, a likely etiological mechanism could be that the direct effect of venom haemorrhagins in the arterial circulation may have caused arterial spasm, vasculitis and subsequent thrombosis.<sup>11</sup> This mechanism could explain the presence of multiple infarcts in this patient.

A distant possibility is that local envenomation can cause local thrombosis in the venous and arterial circulation of the affected limbs. This thrombus could embolise and cause

multiple infarcts as a shower. As this patient had no other signs of embolization, this is unlikely. Thus, occurrence of infarction is multifactorial and more than one cause could contribute to the disposition of the patient.

The initial poor GCS score which rapidly reversed after the administration of ASV suggests the role of the venom in causing widespread cerebral depression and encephalitis.

The study done by Thomas et al.<sup>12</sup> showed that thrombotic complications due to envenomation were not seen if polyvalent ASV was administered within 6 hours after the bite. Even though the patient received early ASV she developed multiple infarcts with haemorrhage but with continued use of ASV, her neurological manifestations improved.<sup>13</sup>

### Conclusion

It is very unusual to find a case of hemiplegia due to multiple ischaemic infarcts as a result of vasculotoxic envenomation as haemorrhages are commoner than infarcts in viper bites. In this case even though the species of snake was not known, the clinical features were characteristic of a viper bite. The patient even after receiving polyvalent ASV within few hours of bite continued to worsen clinically, as the extent of manifestations in a given patient depends on the amount of venom that was injected and the effectiveness of treatment depends on the ability of the ASV to bind free toxins in the blood.

Hence in snake bite victims with poor GCS, administration and continuation of polyvalent ASV should be based on clinical presentation, haematological parameters and imaging studies rather than a single parameter.

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### PEER REVIEW

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### CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

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### PATIENT CONSENT

The authors, *Bhojaraja MV, Prabhu MM, Stanley W, Sanket S, Marimuthu VKN and Kanakalakshmi ST*, declare that:

1. They have obtained written, informed consent for the publication of the details relating to the patient(s) in this report.
2. All possible steps have been taken to safeguard the identity of the patient(s).

- This submission is compliant with the requirements of local research ethics committees.

Figures 1-6: MRI BRAIN: T2 and FLAIR sequences showing hyper intensities which are s/o acute to sub-acute infarcts

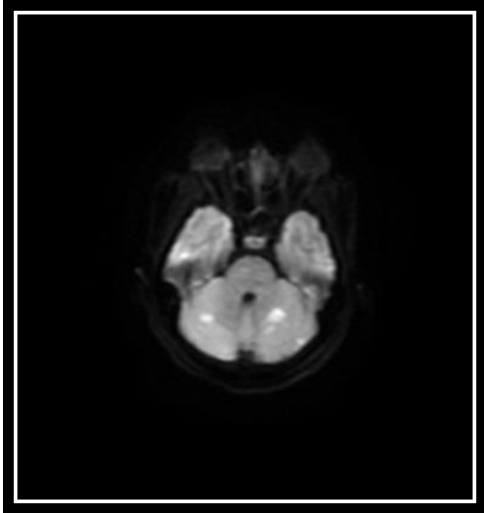


Figure 1

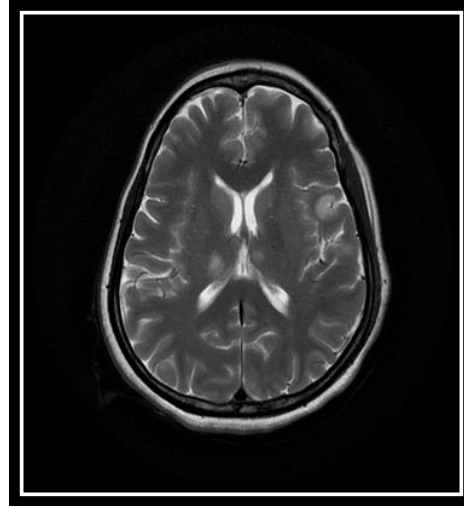


Figure 4

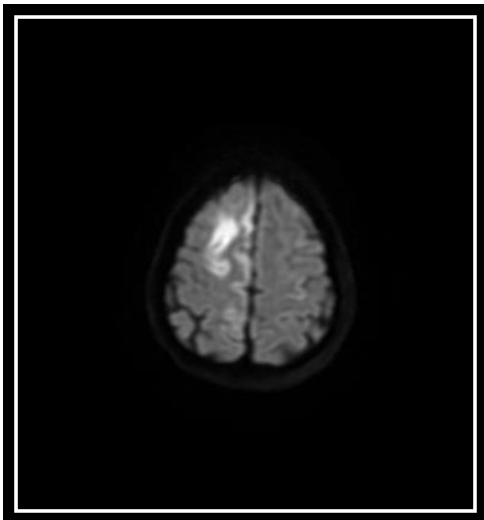


Figure 2

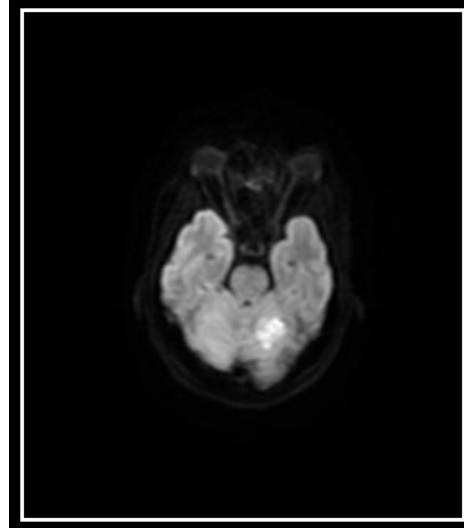


Figure 5

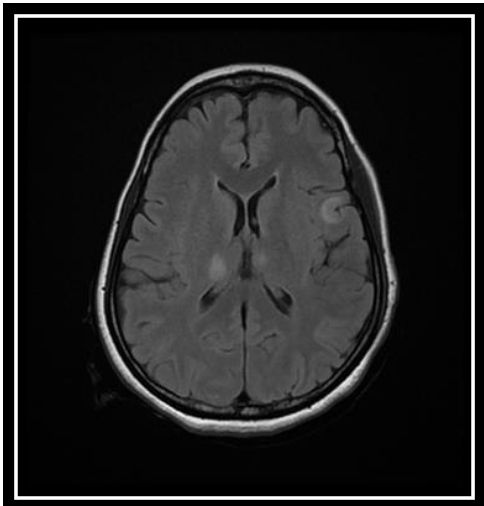


Figure 3

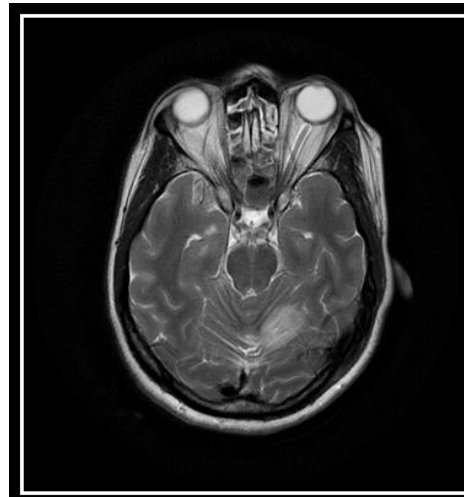


Figure 6