

## Cerebral Infarct with Septicemia: An unusual presentation of Snake Bite

Saha K, Saha D, Mondal RRS, Ranjit P, Sarkar S, Thiyagrajan G

### ABSTRACT

Complications arising from snake bites are often varied and unexpected. The clinical presentations are dictated by the species of snake and the severity of envenomation. Local necrosis, bleeding, acute renal failure and acute respiratory distress syndrome are common manifestations of hematotoxic snake bite. We report a case of rare thrombotic manifestation, secondary to acquired hypercoagulable state in a patient with hematotoxic snake bite. The patient had left middle cerebral artery (MCA) territory infarction, acute renal failure and uremic encephalopathy in post snake bite state, also developed heparin induced thrombocytopenia during repeated dialysis.

**Key words:** cerebral infarct, snake bite, hematotoxic, snake bite

### INTRODUCTION

Annually, more than twenty five lakh snake bites are reported in India, of which 30,000 to 50,000 people die.<sup>1</sup> Common venomous snakes found are cobra (*Naja naja*), Krait (*Bungarus caeruleus*), Russell's viper (*Daboia russelii*) & Saw Scaled Viper (*Echis carinatus*), also known as the 'big four' of India. Their habitat is widely distributed across the country. Other venomous snakes like the King cobra (*Ophiophagus hannah*), Hump Nosed Pit Viper (*Hypnale hypnale*), Banded Krait (*Bungarus fasciatus*) are also found in India. The distribution of these snakes is limited to certain geographic regions. The ophitoxaemia due to viperine species manifest primarily as hematological complications. The clinical characteristics include local cellulitis, renal failure, and hemorrhagic manifestations including pituitary and intracranial hemorrhage. In this report we present an unusual complication, cerebral infarction following snake bite.

### CASE REPORT

A 32 years old man presented 12 hours after an alleged snake bite with excruciating pain, swelling and redness over the right foot and leg. He had a bite mark over the dorsum of the right great toe. The patient had developed aphasia, deviation of

angle of mouth to the left, and weakness of the right half of the body, all of which were sudden in onset, and began around 6 hours following the bite. After the bite, he had passed urine once which was bloody in appearance; and had received 15 vials of polyvalent anti venom serum (AVS) at the local hospital. He was non diabetic, non-hypertensive and had no addictions or significant past history. At admission, the patient was restless and disoriented, with a GCS score of E4M3V2. General examination revealed: Afebrile, pulse 84/min, BP 126/80 mm of Hg, no pallor, icterus, cyanosis, edema or clubbing. The right foot was inflamed and tender to touch. Plantar was extensor on right side and flexor on left, while jerks of the right side were diminished. Other systems were clinically unremarkable. 100 ml of reddish urine was evident on catheterization but no other bleeding manifestations were evident. The whole blood clotting time was 34 minutes. He was put on IV crystalloids and IV antibiotics. There was no improvement on renal output; hence hemodialysis (HD) was planned the next day.

The following report were available the next morning: urea 67 mg/dl, creatinine 3.3 mg/dl, Na+ 137 m eq/L, K+ 5.5 meq/L, Hb 17.3 gm%, WBC 38,600/cu mm, Neutrophils 90% and Lymphocytes 10%, Prothrombin time 24 second, INR 2.0 and APTT 45 sec. The patient underwent HD on the day

following admission. Meanwhile, CSF studies including culture and viral serology were unrewarding. Non contrast CT scan of brain was normal.

Despite ongoing HD, urea and creatinine gradually increased, touching 190 mg/dl and 11.2 mg/dl respectively after 4 days of admission, and worsened with heparin (used during HD) induced thrombocytopenia (HIT), platelet count: 42,000/cu mm, resulting in multiple echymotic patches. Heparin during HD was stopped. The patient had 5 HD sessions on alternate days. From the 6<sup>th</sup> day after admission, the output began to improve and counts, urea and creatinine levels started to drop. However, the patient remained aphasic and the right sided hemiparesis persisted after 10 days of admission. Contrast MRI of brain done on the 11<sup>th</sup> post admission day documented sub-acute infarct in the left MCA territory.



**Fig. 1.** Coronal section shows Temporal (MCA territory) infarction

It was labeled as a case of hemotoxic snake bite presenting with ischemic CVA, followed by coagulopathy and acute kidney injury. A search for established causes of a pro-coagulant state (ANA, VDRL, Coagulation studies and lipids including Lp(a) and homocystiene were non-contributory.

## DISCUSSION

Cerebral complications, particularly ischemic complications, are very rare in viperine

envenomation.<sup>2-4</sup> Cases of pontine infarct, right occipital and bilateral cerebellar infarct had been reported.<sup>5,6</sup> In a study involving 309 patients, the reported rate of cerebrovascular complications was only 2.6%.<sup>2</sup>

The occurrence of infarcts following viper envenomation had several hypotheses. Hypotension was documented as one of the most common causes of infarcts.<sup>7,8</sup> This is due to vasodilatation and loss of vasomotor tone provoked by the viper toxin. Russell's viper toxin has been shown to have both procoagulant and anticoagulant properties.<sup>7,9</sup> Arginine esterase hydrolases present in viper venom are known to aggregate platelets. Activation of procoagulant pathway can lead to intraluminal thrombus formation. As in early disseminated intravascular coagulopathy (DIC), direct action of toxins causes vascular endothelial damage, with probable release of vascular endothelial growth factor and von Willebrand's factor that also promote thrombus formation and subsequent infarcts.<sup>7</sup> Direct cardiotoxic effect of viper venom leading to dysrhythmias, which provoke cardiac thromboembolism, also has been implicated in ischemic sequelae.<sup>8</sup>

A combination of factors could have caused the infarction in our patient. The presence of local inflammation, elevated total leucocyte counts and neutrophilic shift, even though there was no fever, may have been an indication of early sepsis. The cerebral infarction can also be the result of toxic vasculitis or toxin induced vascular spasm and endothelial damage. Our patient, despite treatment with ASV within 1 hour of envenomation developed cerebral infarction.

## CONCLUSION

Cerebral infarction can be one of the differential diagnoses of neurological deterioration following viperine envenomation.

---

**AUTHOR NOTE**

---

**Kaushik Saha**, Assist.Prof.,General Medicine  
Contact no- +919831937270,  
Email: drkaushikmedped@gmail.com  
**(Corresponding Author),**  
Calcutta National Medical College, Kolkata  
**Dipa Saha**, Assist.Prof., Physiology, College of

Medicine & JNM Hospital, Kalyani, West Bengal  
**Rabi Ranjan Sow Mondal**, 2nd year post-graduate  
**Parinita Ranjit**, 3rd year postgraduate  
**Sujoy Sarkar**, 3rd year postgraduate  
**G. Thiyagrajan**, 2nd year postgraduate  
Department of General Medicine, Calcutta  
National Medical College, Kolkata

---

**REFERENCES**

1. David AW. Guidelines for the clinical management of snake-bites in the south-east Asia region. World Health Organization, Regional Office for South East Asia, New Delhi: 2005. p. 1-67.
2. Mosquera A, Idrovo LA, Tafur A, Del Brutto OH. Stroke following Bothrops spp.snakebite. *Neurology* 2003;60:1577-80.
3. Bashir R, Jinkins J. Cerebral infarction in a young female following snake bite. *Stroke* 1985;16(2):328-30.
4. Murthy JM, Kishore LT, Naidu KS. Cerebral infarction after envenomation by viper. *J Comput Assist Tomogr.* 1997 Jan-Feb;21(1):35-7.
5. Lee BC, Hwang SH, Bae JC, Kwon SB. Brainstem infarction following Korean viper bite. *Neurology.* 2001;56(9):1244-5.
6. Mugundhan K, Thruvarutchelvan K, Sivakumar S. Posterior circulation stroke in a young male following snake bite. *J Assoc Physicians India.* 2008;56:713-4.
7. Boviatsis EJ, Kouyialis AT, Papatheodorou G, Gavra M, Korfiyas S, Sakas DE. Multiple hemorrhagic brain infarcts after viper envenomation. *Am J Trop Med Hyg.* 2003;68(2):253-7.
8. Malbranche S, Piercecchi-Marti MD, Thomas L, Barbey C, Courcier D, Bucher B, et al. Fatal diffuse thrombotic microangiopathy after a bite by the "fer-de-lance" pit viper (*Bothrops lanceolatus*) of Martinique. *Am J Trop Med Hyg.* 2008;78(6):856-61.
9. Ameratunga B. Middle cerebral occlusion following Russel's viper bite. *J Trop Med Hyg.* 1972;75(5):95-7.