Anterior Circulation Stroke Following Snakebite: A Rare Presentation

Ciji Sebastine, Neha Athale, Jaishree Ghanekar, Sainath Hegde

ABSTRACT

India is estimated to have the highest snakebite mortality in the world. Most fatalities are due to delay in getting the definitive treatment. Most snakebites are inflicted on the lower limbs of farmers, plantation workers, herdsmen, and hunters in rural areas. The viper is one of India’s most commonly encountered poisonous snakes and envenomation following viper bite usually leads to consumption coagulopathy. Clinical characteristics include cellulites, renal failure, hemorrhagic manifestations including pituitary and intracranial hemorrhage. In the setting of viper envenomation, large-vessel thrombosis is a very rare occurrence. Also, bilateral anterior cerebral artery infarction, when unrelated to anatomical abnormalities, surgery or trauma, itself is an exceedingly rare event. The following case is an unusual one of bilateral cerebral infarction in ACA territory in an otherwise healthy individual.

Abbreviations: ACA: Anterior cerebral artery; ASV: Anti snake venom; CNS: Central nervous system.

Keywords: Stroke, Snakebite, MRI study, Anterior circulation.

How to cite this article: Sebastine C, Athale N, Ghanekar J, Hegde S. Anterior Circulation Stroke Following Snakebite: A Rare Presentation. MGM J Med Sci 2014;1(3):143-145.

Source of support: Nil

Conflict of interest: None

CLINICAL HISTORY

The case was a 48-year-old female in an otherwise healthy condition, presented with alleged history of snakebite two days earlier. Bite was on third finger of left hand associated with history of swelling of left upper limb. There was no diplopia, slurring of speech or respiratory embarrassment or any signs of bleeding manifestations and seizure episodes. Following the bite patient had lost consciousness and was taken to nearby hospital where she received ASV. She recovered consciousness after 2 hours in hospital but the patient had problem in recognizing people. She complained of headache on waking up and had one vomiting episode. By evening the next day, the patient who was conscious oriented by now realized that she could not move the left side of body. The weakness started with involvement of lower limb and gradually involved upper limb over period of few hours and then the patient was transferred to our hospital. There was no history of incontinence of urine.

ON EXAMINATION

The patient was conscious, oriented in time, place and person, with pulse rate of 88/minute regular, good volume. All peripheral pulses present. BP was 130/90 mm in right upper arm in supine position. On local examination there was swelling of left upper limb till arm. Central nervous system examination revealed fully conscious patient with normal speech. All cranial nerves were normal on examination. Motor system examination showed power of grade 2/5 in left upper limb and grade 1/5 in left lower limb with exaggerated tendon reflexes and extensor plantar response on left side. Patient’s Magnetic Resonance Imaging (MRI) was done which revealed bilateral high frontal lobe infarcts and left parahippocampal gyrus (Figs 1A and B). Magnetic Resonance Imaging angiography could not be done due to cost factor.

Routine blood investigations did not reveal any significant abnormality (Table 1).

Examination of fundus did not show any abnormality. ECG and 2D Echo were normal. Patient was treated with tapering doses of Anti Snake Venom, Anticoagulants, Antiplatelets and Low Molecular Weight Heparin. Glycerine MgSO₄ dressing of left upper limb was done. Gradually power on left upper limb and lower limb improved and swelling disappeared over a period of 1 week. Patient was discharged after 10 days with regular follow-up and physiotherapy.

### Table 1: Blood investigations

<table>
<thead>
<tr>
<th>Parameters</th>
<th>Day 1</th>
<th>Day 3</th>
</tr>
</thead>
<tbody>
<tr>
<td>Hemoglobin (gm/dl)</td>
<td>11.6 gm/dl</td>
<td>10.5 gm/dl</td>
</tr>
<tr>
<td>WBC/mm³</td>
<td>12,800/mm³</td>
<td>11,500/mm³</td>
</tr>
<tr>
<td>Platelets/mm³</td>
<td>2.0 lakhs</td>
<td>2.2 lakhs</td>
</tr>
<tr>
<td>Bleeding time</td>
<td>2 mins 15 secs</td>
<td>2 mins 10 secs</td>
</tr>
<tr>
<td>Clotting time</td>
<td>3 mins 45 secs</td>
<td>3 mins 30 secs</td>
</tr>
<tr>
<td>Prothrombin time</td>
<td>14 secs</td>
<td>12 secs</td>
</tr>
<tr>
<td>Serum creatinine (mg%)</td>
<td>1.6 mg%</td>
<td>0.88 mg%</td>
</tr>
<tr>
<td>Sodium (mmol/l)</td>
<td>132 mmol/l</td>
<td>140 mmol/l</td>
</tr>
<tr>
<td>Potassium (mmol/l)</td>
<td>2.7 mmol/l</td>
<td>3.2 mmol/l</td>
</tr>
</tbody>
</table>
DISCUSSION
In India, more than 200000 snakebites are reported annually. The most important species are cobras, common krait and Russell’s viper. An annual snakebite mortality of 30,000 has been recorded.1

Most snakebites are inflicted on the lower limbs of farmers, plantation workers, herdsmen, and hunters in rural areas. The snake is usually trodden at night or in undergrowth. Some enter human dwellings at night and may bite people who roll over on to them while sleeping on the floor. Seasonal peaks in the incidence of snakebite are associated with agricultural activities, such as ploughing before the annual rains. Severe flooding, by concentrating the human and snake populations, has given rise to epidemics of snakebite. Invasion of virgin jungle during construction of new highways and irrigation has led to an increased incidence of snakebite.2

Snakebite can either be vasculotoxic or neurotoxic. Vasculotoxic snakebite usually lead to disseminated intra-vascular coagulation with consumption of clotting factors. Neurological deficits of vasculotoxic snakebite are usually due to intracranial bleeding rather than infarction.

Cerebral complications, particularly ischemic infarction after viper bite is rare3 Ischemic stroke due to infarction in middle cerebral artery territory following viper bite has been reported by few authors.4 Brainstem stroke had been reported following Korean viper bite and envenomation from Bathrops lanceolatus that is found only in Martinique.5 Additionally, the anterior cerebral arteries are infrequent sites of thrombosis, and bilateral infarction in their territories is an exceedingly rare entity.6

Vasculotoxic venom is a mixture of numerous enzymes which have opposing effects. One set of enzymes cause hypofibrinogenemia, hypoprothrombinemia, thrombocytopenia leading to bleeding manifestations. Other enzymes such as potent proteases lead to activation of clotting factors X and V promoting coagulation. Effect of hyaluronidase causes damage to connective tissue leading to enhanced toxin dissemination.

There is activation of intrinsic coagulation pathway leading to formation of numerous new thrombi in circulation which in turn leads to consumption of coagulation factors and platelets which may result in internal and external bleeding. Another cause is endothelial injury caused by toxic agents such as hemorrhagins which can lead to hemorrhage.7 Occlusions of arteries due to microthrombi are rare clinical findings.8

There are various theories which could lead to ACA territory bilateral infarct.
1. DIC induced by some snake toxins such as viper, cause vessel occlusive thrombi with an underlying procoagulant state which leads to formation of thrombi.8,9
2. Variations in viper venom composition, in terms of its hemorrhagic, anticoagulant, and other activities, may favor thrombosis, as opposed to bleeding.9
3. Hemorrhagins induces vasospasm in arterioles leads to vasodilatation leading to endothelial damage and increased vascular permeability.
4. Preexisting procoagulant state like mutation in factor V, Protein S and C deficiencies can be another reason. Hyper-viscosity state due to hemoconcentration can also be one of the reasons.10
5. Hypotension due to hypovolemia from sweating, vomiting, low blood flow area leads to water shed infarcts.

CONCLUSION
We report this case to highlight this uncommon presentation of viper bite (as hemiplegia), with bilateral infarcts in territory of anterior cerebral artery. Our patient, despite treatment with ASV within 1 hour of envenomation developed delayed cerebral infarction on the second day. Clotting time was normal which ruled out coagulopathy as a cause. The
possible cause of infarct in the anterior circulation is due to toxic vasculitis caused by injury to the endothelium by snake venom toxin. Our case also illustrates that one should work up for possible cerebral infarction in a victim of viper envenomation and focal deficit.

REFERENCES