CASE REPORT

Myocarditis along with acute ischaemic cerebellar, pontine and lacunar infarction following viper bite

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SUMMARY

Cerebrovascular complications are rare following viper bites. A 65-year-old man presented with loss of consciousness and developed haemiparesis following a viper bite. Coagulation parameters were severely deranged. MRI showed acute ischaemic infarction on the left side in the precentral and postcentral gyrus, hemipons and cerebellum. Troponin T was elevated and transient left bundle branch block was seen. The patient had a good outcome following treatment with Anti Snake Venom and supportive therapy. Possible mechanisms of infarction are discussed.

BACKGROUND

Snake bites are a vastly underreported phenomenon in South East Asia. An estimated 35 000–50 000 people die every year in India due to complications of snake bites.¹ Snake venom is predominantly of two types—haemotoxic and neurotoxic. The Viperidae family of snakes has haemotoxic venom. The most common complications of viper bite envenomation are haemorrhagic complications. Cerebrovascular complications have been reported, although they are uncommon. Acute ischaemic stroke, especially that involving the posterior circulation is exceedingly rare following a viper bite.^{2–5} Myocarditis has not been reported as a consequence of viper envenomation in humans. We report myocarditis along with cerebellar, pontine and lacunar infarcts following a viper bite.

CASE PRESENTATION

A 65-year-old man was found unconscious in a farm, bleeding from the mouth and left ankle. On arrival at the emergency triage, his consciousness had only improved minimally. The history given by

family members was only significant for occasional alcohol intake. He was a non-smoker. His vitals were stable. Fang marks were noted over the left medial malleolus with bleeding from the wound site. Neurological examination revealed a Glasgow Coma Scale score of 10/15 (E-4, V-2, M-4). Pupils were bilaterally equal and reactive. Power was >3/5 in all four limbs, the plantar reflex was flexor bilaterally. Examination of other systems did not reveal any abnormalities. Consciousness improved after admission but the patient was noted to have slurring of speech and right hemiparesis (power 1+/5 in right upper and lower limbs); he also developed cellulitis of the left lower limb in a very short duration of time.

INVESTIGATIONS

Laboratory reports were significant for elevated white cell count (17 900/mm³) and a deranged coagulation profile (prothrombin time >120 s). Troponin T was elevated (0.325 ng/mL), as was the serum urea (88 mg/dL). An ECG was performed which showed features of a left bundle branch block (figure 1). An echocardiogram was performed and it showed septal beaking suggestive of a left bundle branch block. There were no regional wall motion abnormalities and no mitral regurgitation. A CT of the brain did not show any abnormalities. An MRI of the brain revealed acute ischaemic infarction on the left side in the precentral and postcentral gyrus, hemipons and cerebellum (figures 2–7).

TREATMENT

He was started on equine polyvalent Anti Snake Venom (Bharat Serum and Vaccines Ltd.

Figure 1 ECG on admission.

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Figure 2 Axial diffusion-weighted image section of the left cerebellum.

ASV—ASIA, Thane, India) immediately. He was also given ceftriaxone and clindamycin alongside supportive therapy. A total of four units of fresh frozen plasma were infused. A magnesium sulfate dressing was applied to the left lower limb. The coagulation profile began to normalise soon after starting the Anti Snake Venom—the international normalised ratio had come down to 1.15 by day 3 of admission. The left bundle branch block also resolved (figure 8). The patient began physiotherapy for the affected side. Limb power on the affected side improved from 1+/5 after admission to 4+/5 at the time of discharge 12 days later.

OUTCOME AND FOLLOW-UP

The patient had improved clinically and symptomatically at the time of discharge. There was some residual lower limb swelling

Figure 4 Axial diffusion-weighted image section of the left hemipons.

on follow-up 1 month later. He was asymptomatic on review 2 months later.

DISCUSSION

Acute ischaemic stroke following viper bite is rare but well documented. Del Brutto and Del Brutto⁶ in their review of neurological complications of snake bites found 23 documented cases of cerebral infarction following a viper bite. An overwhelming majority of the cases reported cortical infarcts, with only one case of brainstem infarction. Gouda *et al*³ and Gupta *et al*⁴ reported two different cases of cerebellar infarction following viper bite. However, this patient developed infarcts in two different arterial territories, raising questions about the possible aetiopathogenesis. Among the various theories postulated to explain ischaemic cerebral infarction following viper bite are

Figure 3 Axial T2 fluid-attenuated inversion recovery section of the left cerebellum.

Figure 5 Axial T2 fluid-attenuated inversion recovery section of the left hemipons.

Figure 6 Axial diffusion-weighted image section of the left cerebral cortex.

Figure 7 Axial T2 fluid-attenuated inversion recovery section of the left cerebral cortex.

hypotension,⁷ hypercoagulopathy⁷⁻⁹ and vasculitis due to endothelial damage.⁷⁻¹⁰ In this case, hypotension was an unlikely cause since the location of the infarct was not in a watershed area. Vasculitis due to the effect of the haemorrhagin toxin on the endothelium could explain the stroke but the short duration in which the infarction developed and the unilateral incidence does not fully support this theory,⁷ although pre-existing vulnerabilities may have existed in the affected regions making them more susceptible to infarction. Hypercoagulopathy due to pro-coagulant factors and/or disseminated intravascular coagulation is supported by the deranged coagulation profile.

While acute myocardial infarction following viper bite has been well documented, myocarditis following viper envenomation has only been reported in animals—in dogs¹¹⁻¹² and in two horses that were injected with venom for antivenom production.¹³ Myocarditis is hypothesised to occur either due to direct effects of the toxin on the myocardium⁹⁻¹⁰ or hypersensitivity to the venom.¹⁴ The patient's elevated troponin levels

support the diagnosis of myocarditis¹⁴ especially since the ECG and the echocardiogram showed no features to suggest a myocardial infarction. The resolution of the bundle branch block following treatment with Anti Snake Venom indicates that the toxin could indeed have been responsible for the cardiac pathology in this patient.

Previously reported cases of posterior circulation infarction following snake bite have usually resulted in fatalities. In one case the patient survived but had no improvement in neurological status and was given poor prognosis.⁵ In the other case the patient recovered significantly.³ The quick identification of envenomation, attributing the haemiparesis to the effects of snake venom and the immediate institution of Anti Snake Venom therapy along with supportive therapy led to a wholly positive outcome for this patient. This case underlines the importance of identifying uncommon complications of poisonous snake bites and the timely use of Anti Snake Venom.

Figure 8 ECG on day 3.

Learning points

- ▶ Acute ischaemic stroke is a very rare complication of viper envenomation, which usually causes haemorrhagic manifestations.
- ▶ The exact mechanism of cerebral ischaemia and myocarditis is not yet known but direct toxic effects of the venom on the endothelium and myocardium could be responsible.
- ▶ This case shows the importance of initiating Anti Snake Venom therapy as soon as signs of envenomation are noted.

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Competing interests None.

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