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Case Report

An uncommon case of venomous snake bite complicated by intracranial haemorrhage

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ABSTRACT

Snake bite is a widespread and neglected public health problem in tropical and subtropical countries, where rural populations are mainly affected. Although venomous snake bite may lead to a wide range of complications, life-threatening neurological complications such as Intracerebral Haemorrhage (ICH) are rare and infrequently documented in literature. Here we discuss the case of a 64-year-old man who developed ICH following a viper bite, and the neurological sequelae that followed after management.

Keywords: Intracerebral hemorrhage, Neurological sequalae, Snake bite, Viper bite

INTRODUCTION

Snake bite is a widespread public health problem in tropical and subtropical countries, where rural populations are mainly affected. It is a common occupational hazard seen mainly in farmers, plantation workers, herders and laborers leading to significant morbidity and mortality that remains largely unreported. India is reported to have the highest number of snake bites (approximately 25,00,000) and snake bite induced mortality (30,000-50,000) per year.[1] Although venomous snake bite may lead to a wide range of complications, life-threatening neurological complications such as Intracerebral Infarction/ Haemorrhage (ICH) are rare and infrequently documented in literature.[2,3]

CASE REPORT

A 64 year old male presented to the emergency with an alleged history of snake bite on the right foot about two hours prior to presentation. He collapsed soon after the bite and was brought to the hospital in an unconscious state. There were no systemic symptoms on presentation and no local or systemic bleeding manifestations. On examination, the vitals were stable, but there was no response to verbal or painful stimuli. The pupils were reacting normally to light; both the conjunctiva showing signs of congestion. The patient was moving all four limbs spontaneously, with restricted movements on the left half of the body. There was a tourniquet at the right thigh with edema of the right leg and foot, and visible fang marks. The systemic examination was normal, with no evidence of bleeding from any site. The oxygen saturation levels were normal with no respiratory distress, and the urine output was normal with one episode of hematuria.

Most of the routine investigations were within normal limits, except for a deranged coagulation profile. Thrombocytopenia was observed along with suspicion of Disseminated Intravascular Coagulation due to elevated Prothrombin Time/International Normalized Ratio (PT/INR), Activated Partial Thromboplastin Time (aPTT), D-dimer levels and Fibrinogen Degradation Products (FDPs). Indirect bilirubin levels were raised, along with Lactate Dehydrogenase (LDH) levels (Table 1). The peripheral smear showed immature and

fragmented RBCs, which was suggestive of hemolytic anaemia. A Non-Contrast Computed Tomography (NCCT) scan of the brain was done immediately, which was suggestive of a small right intracranial bleed without any peri-lesional edema or midline shift (Figure 1).

Table 1: Coagulation profile of the patient over the course of hospital stay.

Day	PT (seconds)	INR	aPTT (seconds)	Platelets (/mm^3)
Admission day	35.2	2.62	85.4	52,000
Day 2	18.4	1.63	32.8	78,000
Day 3	12.6	1.24	27.6	1,13,000
Day 4	11.2	1.10	26.8	1,48,000

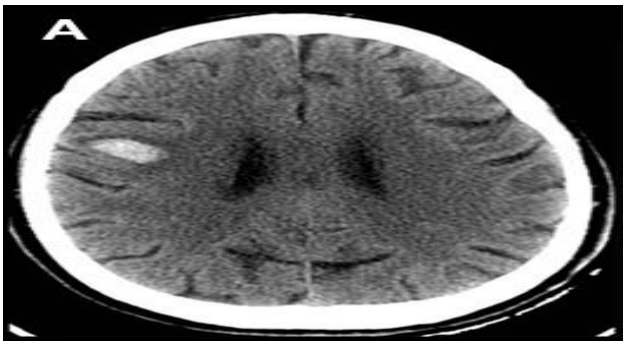

Figure 1: NCCT Brain on first day of admission showing a small right intracranial bleed.

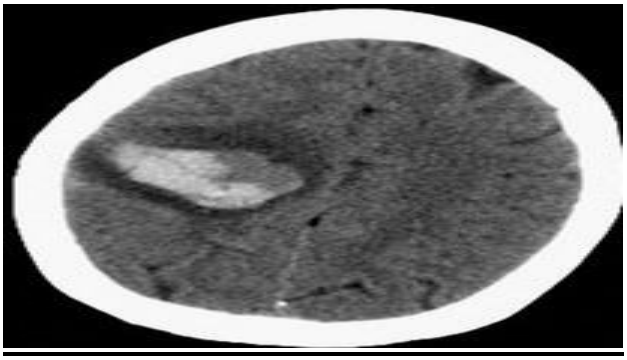

Figure 2: NCCT Brain on second day suggestive of large right fronto-temporal bleed with peri-lesional edema.

The tourniquet was removed on presentation, and the limb was kept immobilized and elevated. Treatment was started with Anti-Snake Venom (ASV) according to standard protocol, after the necessary sensitivity testing. Fresh Frozen Plasma (FFP) was given in view of the deranged coagulation profile, and supportive care was provided with intravenous fluids. In view of the altered consciousness, a foley's catheter was inserted and a feeding ryle's tube was put to prevent aspiration. The next day, the patient had a single episode of generalized tonic clonic seizure for which necessary anti-epileptics were given promptly. A repeat NCCT Brain was done which showed an intra-parenchymal intracerebral hemorrhagic lesion in the right fronto-temporal region, along with surrounding peri-lesional edema (Figure 2). Intravenous Mannitol was administered immediately and a Neurosurgery consultation was sought. Conservative management was advised by the Neurosurgeon, along with continuation of ASV and supportive therapy. The patient regained consciousness on the fourth day of admission, with stable vitals and routine blood parameters. Except for the facial deviation and residual hemiparesis on the left half of his body, the patient showed no major neurological sequelae. His coagulation profile showed marked improvement and he was discharged after two days on anti-epileptics and oral glycerol.

DISCUSSION

Snake bite is a common medical emergency, where timely treatment can reduce morbidity and mortality and save precious human lives. Of the total snake bites, roughly 15% are due to poisonous snakes, of which Viperidae is the largest family.[4] Although occurrence of cerebrovascular complications is quite uncommon following a snake bite, it has been described previously in some case reports.[5,6]

The mechanism of coagulopathy following snake envenomation has not been clearly elucidated. The most common coagulation abnormality associated with snake-bite envenoming is Venom Induced Consumptive Coagulopathy (VICC). Viperine venom has different concentrations of enzymes that include proteases, phospholipase A2, hyaluronidase and arginine ester hydrolase. These enzymes cause haemolysis secondary to red cell membrane damage, and promote muscle necrosis.[7] There is formation of weak fibrin clots due to the combined action of these enzymes and the other thrombogenic enzymes, which inturn activate plasmin and result in consumption coagulopathy and haemorrhagic consequences.[8] Snake venom also contains prothrombin activators which cause a variable deficiency in factor V, VII and fibrinogen.[9] This combined deficiency of clotting factors leads to DIC, which is responsible for spontaneous bleeding tendencies as evidenced by the intracranial hemorrhage seen in our case.

Treatment should be aimed at stopping the progression of bleeding, and preventing consumption coagulopathy. ASV is the definitive treatment of poisonous snake-bites and it helps in neutralizing the circulating toxin. 100ml of polyvalent ASV is to be given initially after skin testing for systemic envenomation in Viperidae family, followed by a repeat dose of 100 ml one to two hours after the first dose if there is no clinical or neurological improvement. It is to be continued in patients with persistence or recurrence of blood incoagulability after six hours of first

dose. The use of FFP has been seen to be associated with faster recovery and reduced risk of bleeding.[7,10]

CONCLUSION

Venomous snake bite can present with an uncommon but potentially disastrous complication in the form of intracerebral haemorrhage. Early diagnosis after presentation, rapid administration of ASV, surgical decompression as indicated, and supportive treatment with FFP and blood products can substantially improve the patient outcome in the majority of cases.

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