A Rare Snake Bite Sequelae: Intracerebral Haemorrhage with Cerebellar Infarction

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ABSTRACT

Introduction: India is a nation referred by the western populace, as a nation of snake charmers over hundreds of years. The people at danger of snakebite in our nation is around 50 million. Every year, more than 2,500,000 snake bites are accounted for in India, out of which around 50,000 cases end in mortality.

Case Report: Here is a case of 80-year-old female patient, with history of snake bite over left foot presenting with irritability and Glasgow coma scale of 13/15, with power of left upper and lower limb 1/5, increased tone and extensor plantar with MRI suggestive of subacute intraparenchymal and subarachnoid haemorrhage without midline shift along with acute infarct in the territory of posterior-inferior cerebellar artery.

Conclusion: Cerebral complications arising due to snake venom, includes infarcts and haemorrhages. Prompt aggressive approach towards coagulation defects and appropriate supportive measures results in better outcomes in cerebral complications.

Keywords: Snake bite, Intraparenchymal and sub-arachnoid Haemorrhage.

INTRODUCTION

India is a nation referred by the western populace, as a nation of snake charmers over hundreds of years. People at danger of a snakebite in our nation are around 50 million individuals. Every year, more than 2,500,000 snake bites are accounted for in India, out of which around 50,000 cases end in mortality.[1]

Ophitoxaemia (snake bite envenomation) can give cellulitis, renal complications and haemorrhages including intracranial haemorrhage. Here, we report a bizarre entanglement of Intraparenchymal and subarachnoid bleed occurring after a snake bite.

CASE REPORT

A 80-year-old female patient presented with history of snake bite over left foot, while working in the farm. On examination, the patient was irritable, with a Glasgow coma score of 13/15, power of left upper and lower limb 1/5, increased tone and extensor plantar, and right upper and lower limb being normal. Her swelling gradually extended up to the knee and ecchymosis increased with increment in cellulitis. Eventually, venous Doppler of the affected side was done to rule out to venous thrombo-embolism in view of extended swelling and oedema associated with rise in temperature and pain, which turned out to be normal.

Her pupils were bilaterally equal and reactive to light. General examination revealed large ecchymosis over left leg medial malleolus with diffuse skin involvement. The fang marks were seen close to left medial malleolus. (Fig.1). The coagulation profile was deranged (prothrombin time: 14.9 seconds, International Normalised Ratio : 1.25).

Findings of initial computed tomographic (CT) scan of the brain are shown in figure-2. Magnetic Resonance Imaging (MRI) brain was repeated after 4 days.

The patient was initially treated with anti-snake venom (Total 20 vials—200 mL of anti-snake venom diluted in 200 mL of 0.9% saline over 2 hours) after abnormal 20 min whole blood clotting test. [1 mL of antisnake venom kills 0.60 mg of cobra venom, 0.45 mg of krait venom, 0.6 mg of Russell snake venom, and 0.45 mg of saw-scaled snake venom.]

The coagulation profile was rectified with platelet concentrates and fresh frozen plasma transfusions. Local debridement was done and necessary dressing was given and IV antibiotics were initiated.

Patient improved steadily. Currently, she is conscious, oriented and ambulant with minimal focal neurological deficit.

DISCUSSION

Ophitoxaemia is fairly a colourful term that describes the clinical spectrum of snake bite envenomation. In India, Maharashtra has the most astounding incidence of snake bites (70 bites for each lakh populace), trailed by Kerala, Tamil Nadu, Uttar Pradesh, and West Bengal. Mortality associated with snake bite is related with renal complication, focal sensory system haemorrhage, and auxiliary disease. Cerebral complexities after snake bite envenomation are uncommon and it incorporates infarcts and cerebral haemorrhages.[2,3]

Numerous systems have been proposed for cerebral haemorrhages following snake bite, of which the most widely recognized is venom-actuated consumptive coagulopathy and haemorrhagin-induced direct endothelial damage. Cerebral haemorrhages have been connected to the bites of venomous snakes

In an investigation of 309 patients with snake bite, Mosquera et al announced cerebrovascular complications

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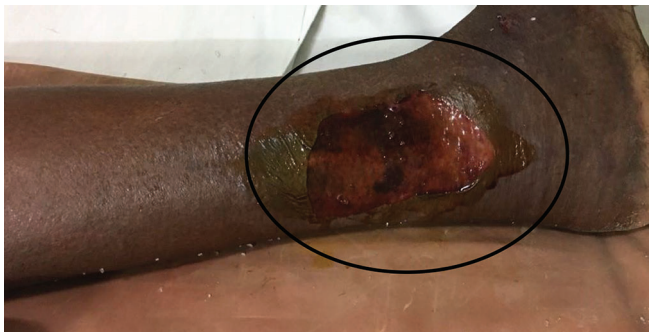

Figure-1: Extensive ecchymosis over left medial aspect of leg near medial malleolus with diffuse skin loss near wound area

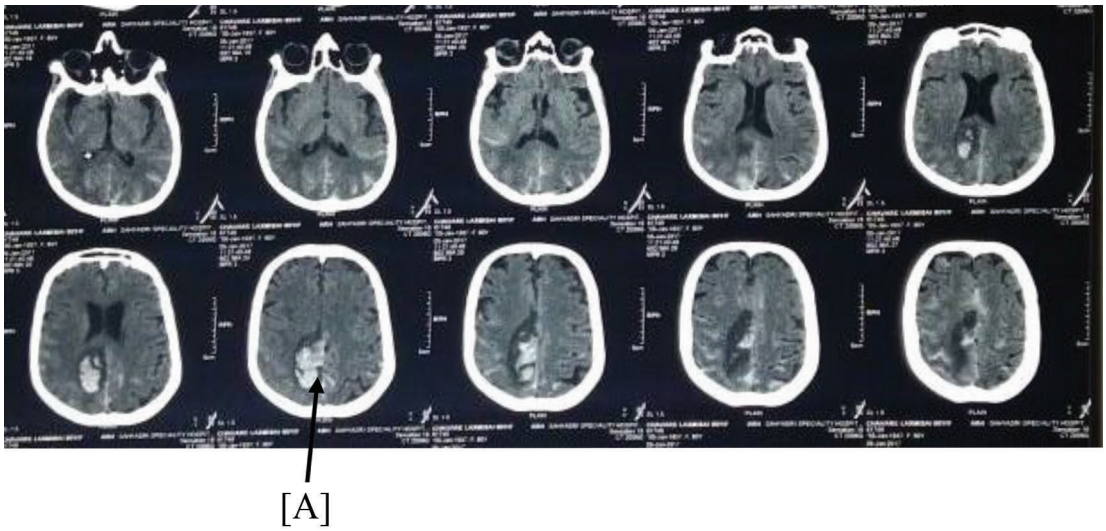

Figure-2: CT brain. [A] Haemorrhage in right parietal, occipital sulcus, percuneus, cuneus, centrum semiovale and high parietal region.

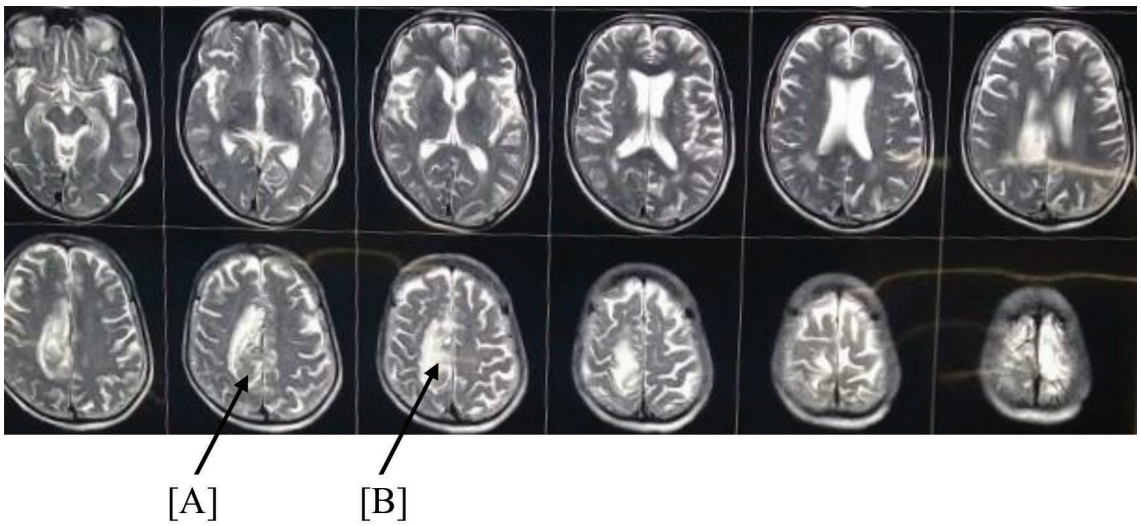

Figure-3: MRI Brain. [A] acute infarct in territory of left posterior, inferior cerebellar artery on left cerebellar hemisphere. [B] large Intraparenchymal haemorrhage with blood products in early to late subacute stage involving cingulate gyrus, percuneus and centrum semiovale on right side (vol ~ 16 cc) with subarachnoid haemorrhage diffusely involving the parietal, temporal and occipital spaces bilaterally with no evidence of midline shift.

Snake	Toxins	Effect on brain
Bothrops species	Aspercitin, haemorrhaging, metalloproteinases	Subarachnoid and parenchymal haemorrhages
Daboia russelli (Russell viper)	Proteases	Pituitary haemorrhages
Pseudonaja textilis (Brown snake)	Prothrombinase	Parenchymal brain haemorrhages
Notechis scutatus (Tiger snake)	Toxic acidic proteins	Parenchymal brain haemorrhages

Table-1: Cerebral haemorrhages linked to the bite of venomous snakes

in just 8 patients (2.6%) of which 7 patients had cerebral haemorrhages.[4] Coagulopathy and intracerebral bleed was reported in a 85-year-old lady assaulted by a serpent of Elapidae family (Notechis scutatus).[5]

Snake venom contains proteases, phospholipases, collagenases, metalloproteinases, and thrombin-like enzymes.[6] These interfere with normal clotting of blood. They produce anticoagulant and coagulant impacts. The coagulant impact is because of presence of the arginine esterase hydrolase, which has a comparative activity of thrombin on platelet coagulation. Snake venom additionally contains an element X activator, which causes coagulopathy by platelet aggregation hindrance.

Venom-induced consumptive coagulopathy (VICC) occurs because of activation of the coagulation pathway at different levels by procoagulant poisons. VICC is measured by a drawn out 20-minute entire blood thickening test, prothrombin time, and enacted fractional thromboplastin time. VICC additionally causes an increment in the fibrinogen degradation products.[7]

Metalloproteinases (ecarin and carinactivase) are prothrombin activators which act by decreasing the levels of fibrinogen, component V, and figure VII resulting haemorrhages including cerebral haemorrhages. Discharging poison causes VICC and also guides endothelial harm which can prompt fatal intracerebral bleed, for example-subarachnoid haemorrhage.[7] Proteases annihilate the walls of the veins, enact fibrinolysis, and cause serious haemorrhages.

Prothrombinase complex in snake venom is made of protease factor (f) Xa and cofactor (f) Va. These factors convert prothrombin to thrombin creating coagulopathy, which can bring about parenchymal haemorrhages.

CONCLUSION

Cerebral complications after snake bite envenomation includes infarcts and haemorrhages. Despite the fact that it is uncommon in occurrence, cerebral complications after snake bites are related with high morbidity and mortality. The patient described here had a huge subacute Intraparenchymal and subarachnoid bleed with no confirmation of midline shifts with acute infarct in territory of left postero-inferior cerebellar artery after a snake bite. Prompt treatment with forceful amendment of the coagulation issue and suitable steady measures can bring about a superior result in patients having cerebral entanglements of snake bite envenomation.

REFERENCES

1. David AW. Guidelines for the Clinical Management of Snake-Bites in the South-East Asia Region. New Delhi, India: World Health Organization. Journal of The Association of Physicians of India 2005;64:1–67.
2. Boyer LV, Seifert SA, Clark RF, et al. Recurrent and persistent coagulopathy following pit viper envenomation. Arch Intern Med 1999;159:706–710
3. Murthy JM, Kishore LT, Naidu KS. Cerebral infarction after envenomation by viper. J Comput Assist Tomogr 1997;21: 35–37
4. Mosquera A, Idrovo LA, Tafur A, Del Brutto OH. Stroke following Bothrops spp. snakebite. Neurology 2003;60:1577–1580.
5. Yap CH, Ihle BU. Coagulopathy after snake envenomation. Neurology 2003;61:1788.
6. Cortelazzo A, Guerranti R, Bini L, et al. Effects of snake venom proteases on human fibrinogen chains. Blood Transfus 2010;8:s120–s125.
7. Isbister GK. Snakebite doesn't cause disseminated intravascular coagulation: coagulopathy and thrombotic microangiopathy in snake envenoming. Semin Thromb Hemost 2010;36: 444–451.

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