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Case Report

A Case of Saw Scale Viper Snake Bite Presenting as Intraparenchymal Haemorrhage: Case Report

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ABSTRACT

Snake bite envenomation is a public health hazard in tropical countries. Viperine snake bite may be noxious or non toxic but it may lead to life threatening neurological complications such as intraparenchymal bleeding. Antivenom serum and fresh frozen plasma along with other supportive management may obviate the need for any neurosurgical intervention.

Key Words **-** Saw Scale Viper, snakebite envenomation, tropical disease, intraparenchymal bleed, Antivenom Serum, Fresh frozen plasma.

INTRODUCTION

Snake bite envenomation is a neglected tropical disease affecting millions of people living in the developing world. It is particularly common in the South Asian subcontinent. [1] According to the snake species, the clinical picture may be dominated by swelling and soft tissue necrosis in the bitten limb or by systemic (neurological, cardiac, renal and haematological) manifestations. Serious neurological complications include stroke and muscle paralysis. These are related to the toxic effects of the venom which affects the coagulation cascade, the neuromuscular transmission or both. Metalloproteinases, serine proteases, and C-type lentins (common in viper and colubrid venoms) have anticoagulant or procoagulant activity and may be either agonists or antagonists of platelet aggregation. This results in ischemic or hemorrhagic strokes. Various bleeding manifestations are commonly seen with viperine snake bite. Common bleeding manifestations include bleeding gums, epistaxis, haemoptysis, haematuria, and haematemesis. [2] Amongst the enclosed spaces, bleeding in retroperitoneal space and intracranial bleeding are the most frequent. Haemorrhage in the pleuro-pericardial space is extremely rare. [3] Though subspecies of saw scaled viper snake is considered to be non lethal venomous snake it may amount to rare but potentially life threatening complication like intracerebral bleeding. Here, we report a case of viperine (Echis carinatus) snake bite presenting with intraparenchymal haemorrhage.

CASE REPORT

A 22 year old male, from a village, presented to the casualty department of our hospital with history of six days old snake bite on the right foot. He did not take any treatment for four days considering it to be non noxious. He later developed local site swelling and edema which were progressive in nature.

He was brought to the hospital with breathlessness, altered sensorium and gross cellulitic changes over right foot .On examination, the patient was drowsy, tachypneic, localizing painful stimuli, talking irrelevantly (Glasgow Coma Scale 8). Ophthalmoscopic findings were normal. His pulse rate was 110 per minute, blood pressure was 126/68 mm of Hg. He was intubated and kept on ventilatory support.

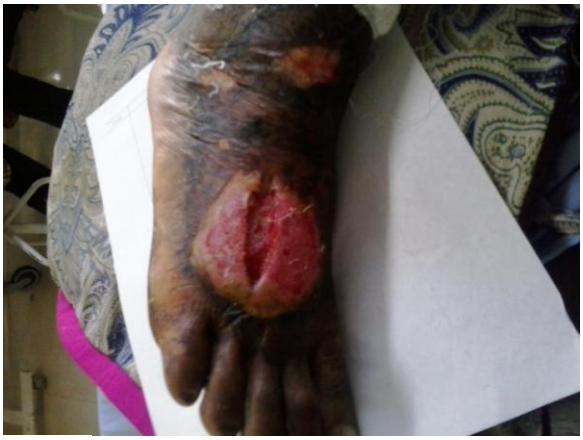

Figure 1 (Fasciotomy for the cellulitis (caused by viperine snake bite) of the right foot)

Laboratory investigations revealed a haemoglobin of 7.5 gm/dl, total leukocyte count of 15,500/mm3, platelet counts 1.8 lacs/mm3.Peripheral blood smear showed normocytic normochromic RBC,mild anisopoikilocytosis,tear drop elliptocytes & schistocytes. Urine analysis showed normal study. Coagulation profile showed bleeding time-3 minutes (normal – upto 9 minutes), clotting time-6'30 minutes (normal – upto 8 minutes),prothrombin time (PT) 16 seconds (Control = 14seconds) with INR 1.11, partial thromboplastin time (PTT) was 34 seconds (Control = 35 seconds),and fibrinogen degradation products - 2.68 mg/L (Normal < 0.20 mg/L), CPK 3111(normal – up to 171) and LDH of 410(normal up to 248). All the biochemical investigations including plasma glucose, blood urea, serum creatinine, serum bilirubin, AST, ALT, serum total protein, and albumin/globulin ratio were within normal limits.

His CT scan brain showed a moderate (58x35 mm) intraparenchymal hemorrhage in left high parietal region with perilesional edema and seepage of blood in occipital horn of both lateral ventricles. There was minimal midline shift towards right. Chest X ray and ECG reports were within normal limits.

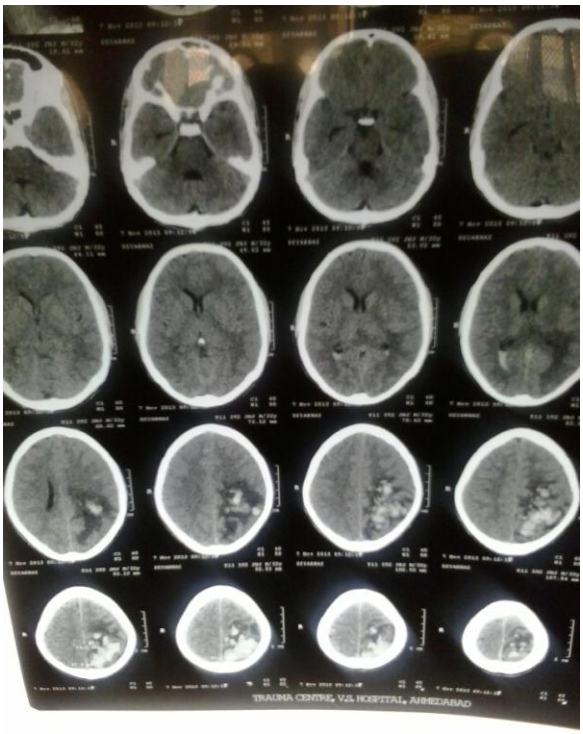

Figure 2. (CT scan brain showing a moderate (58x35 mm) intraparenchymal hemorrhage in left high parietal region with perilesional edema and seepage of blood in occipital horn of both lateral ventricles)

He was treated immediately with 10 vials of polyvalent antivenom serum (AVS) {Vins Bioproduct}, raised against Indian *Daboia russelii*, *Echis carinatus*,

Naja naja and *Bungarus caeruleus* venoms. Each vial was dissolved in 10 ml of sterile water and diluted with 200 ml of normal saline to a total volume of 300 ml and was infused intravenously over an hour to restore the coagulability. He was transfused with platelets and fresh blood to restore normal blood counts. The patient had fasciotomy and stringent sterile dressing for the cellulitis of the foot. He was further managed conservatively as his condition gradually improved. Repeat CT scan showed no increase in size of hematoma or midline shift.So we decided not to intervene neurosurgically. He was weaned from ventilator on the fourth day and later discharged on the tenth day.

DISCUSSION

Snake envenomation is a neglected tropical health hazard. Viper species are most commonly involved in these cases. It mainly affects rural men engaged in agriculture in tropical countries. In the subcontinent of Asia it has been reported that 4 million snake bites occur annually out of which 50% are envenomed leading to 100000 deaths. The degree of severity depends upon the a)size and species of snake, b)amount and degree of toxicity, c)location of bite, d)first aid measures, e)timing of treatment, f)presence of co-morbidity and g) unique susceptibility of the person. Echis carinatus is associated with a mortality rate of 10-20%, if effective treatment is not initiated early. [4,5]

The major cause of mortality is due to increased bleeding tendency caused by the venom. Venom of viper is a complex mixture of biological protein and non protein toxins leading to activation of coagulation pathways and ultimately consumptive coagulapathy.It also contains activators (ecarin and carina activase) of prothrombin causing variable deficiency of fibrinogen,factor V and VII. Vascular endothelial damage caused by the haemorrhagin present in the venom also contributes to bleeding manifestations. Factor X activator and platelet aggregation inhibitors causes marked prolongation of PT, PTTK and FDP. The culmination of the above mechanism leads to disseminated intravascular coagulation. [6,7] In our patient, low platelet count, marginal increase of prothrombin time, and a marked increase in fibrinogen degradation products suggested disseminated intravascular coagulation (DIC). Venom induced consumptive coagulopathy and vascular endothelial damage may have been the putative mechanism of intraparenchymal bleed leading to altered sensorium.

Snake bites may have varied outcomes. Neurodeficits following viper snake bite is not uncommon and it may present as subarachnoid or intraparenchymal bleeding and cerebral infarction. [8,9] Singh S et al found acute flaccid paraplegia in a young female following DIC due to dorsal spinal cord involvement. [10] Right hemiplegia with expressive aphasia in a 18 year old as a result of infarct in middle cerebral artery region was seen by Narang SK et al. [11] Our patient had an intraparenchymal bleed in left parietal region which had a delayed presentation of disorientation.

Antivenom serum treatment given at the earliest significantly bind with the circulating venom reducing the cascade of bleeding mechanism. Fonsenka LC et al reported a case of viper bitten adult where AVS was not effective. [12] They inferred that this may have been due to inadequate dose or ineffective functioning of AVS. Similiarly 167 cases of viper envenomation were studied by Isbister GK et al in Australia. [13] They recommended that Fresh frozen plasma replacement induces faster recovery and decreases the risk of bleeding(by increasing fibrinogen,factorV

and VII).In our case the manifestation of neurological effect was delayed. AVS,FFP and other supportive measures were given simultaneously so it was difficult to judge which therapy worked. But the above treatment helped the patient recover without any neurosurgical intervention. Though AVS decreases the venom load but the cascade of bleeding could be arrested by FFP and other supportive measures.

CONCLUSION

Proper management of the envenomed patient, including prompt transport to the hospital, correction of the hemostatic disorder, ventilatory support, and administration of antivenom, significantly reduces the risk of neurological complications which, in turn, reduce the mortality and improve the functional outcome of survivors. Here we intended to bring about awareness of the delayed but fatal (intraparenchymal haemorrhage) consequences of viper snake bite.

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