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Case report

Spontaneous intracerebral bleed – Snake envenomation

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Abstract

Snake bite envenomation is a commonly encountered emergency in tropical countries like India, with potentially fatal complications. Life threatening neurological complications is rare and infrequently documented in literature. We discuss the case of 28-year-old gentleman, managed successfully for an intracerebral hemorrhage following a viper bite and attempt to obviate some management dilemmas often encountered in viperine envenomation.

Key words: 20 minutes whole blood clot test, anti snake venom, Snake bite envenomation, viper bite

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Venomous snake bite is an important public health hazard in tropical and subtropical countries[1,2]. In India, there are about 216 of identifiable snakes in which 52 are known to be venomous and the major families of poisonous snakes are Elapidae (Cobra, Naga Naga, King cobra & Kraits), Viperidae (Russell`s Viper, Echiscarinatus or saw scaled viper) and Hydropidae (sea snakes)[3].

Venomous secretion is more during warmer months than in cold seasons in all venomous snakes. So, the high fatality rate is seen during in summer and August, September and October months due to high environmental temperature.

Most snakes inject 10% of the available venom in a single strike except Russel`s viper which injects 75% of the venom in one bite and is responsible for high morbidity and mortality in India[4].

Case report

A 28-year-old male patient presented to the triage in altered sensorium with an alleged history of snake bite. His friends identified the snake to be a 'Viper'. Patient was brought to the hospital within 3 hours of snake bite.

Patient was conscious but irritable moving all four limbs spontaneously. He had hematemesis, hematuria (Fig 1), bleeding from bite site. Both his conjunctiva appeared congested. Ptosis is evidently seen. No signs of central or peripheral cyanosis or compartment syndrome. His pupils were equal and reacting well and there were no cerebral localizing signs in the form of hemiparesis or plegia at the time of admission.

His vitals on 09-02-2018 i.e., at the time of admission were pulse rate–102/ min, regular; blood pressure–90/60 mm Hg, SpO_2 –99% at room air, GRBS-84 mg/dl, temperature–98.4^{o}F. His GCS was 15 ($E_4V_5M_6$).

His 20 minutes Whole Blood Clotting Time at the time of admission was 60 minutes.

A plain computed tomographic scan of the brain (CT brain) was done, which was normal at the time of admission.

Fig 1. Urinary bag showing hematuria

The investigations revealed thrombocytopenia with elevated Prothrombin Time/International Normalized Ratio (PT/INR), Activated Partial Thromboplastin Time (aPTT). Other hematological investigations revealed an elevated reticulocyte count, elevated indirect and total bilirubin with raised Lactate Dehydrogenase (LDH) levels, suggestive of a hemolytic anemia. His creatinine levels showed elevations within 24 hours of admission, alarming of an impending renal failure. The values are as detailed below.

1. CBP on 10-02-2018: Hb-15.5 g/dl, TLC–19300, DLC–$N_{84}L_{11}E_4M_1$, Platelets–1.37 lakhs/cumm, Serum creatinine-3.1mg/dl, PT-42 sec (13.6 control), INR–3.08, aPTT-84 sec.
2. 13-02-2018: Hb-5.5 g/dl, reticulocyte count-3%, LFT-Bilirubin 4.5 (direct-1, indirect-3.5), LDH-300 mg/dl, Serum creatinine–4.1 mg/dl. Initiated on heparin free dialysis. The peripheral smear showed immature RBCs, spherocytes with reduced platelets, fragmented RBCs and polychromasia, suggestive of hemolytic anemia.
3. 14-02-2018: Chest X-ray revealed diffuse infiltrates, suggestive of ARDS
4. 15-02-2018: Serum Creatinine-9.6 mg/dl
5. 16-02-2018: Serum Creatinine-6.6mg/dl
6. 19-02-2018: PT-25 sec, INR-1.83, Serum Creatinine- 8.7mg/dl, Hb-11.7 gm/dl, Platelets-38000/cumm

He was started on Anti Snake Venom (ASV), initially with 20 vials as WBCT was >60 minutes. WBCT was repeated every 6 hour, 10 vials of ASV administered each time till WBCTwas <20 minutes. A total of 80 vials were administered to reduce WBCT to <20 minutes.

Hemodynamic support included IV fluids, 4 units of Fresh frozen plasma, 4 units of platelet concentrates, 4 units of packed cells.

Antibiotics (Inj. Meropenem-500mg IV BD, Inj. Augmentin-1.2 gm IV BD), diuretics (Inj. Lasix 60 mg IV BD), anti-hypertensives (Tab. Cilnidipine 10 mg BD), gastric ulcer prophylaxis (Inj. Pantop 40mg IV OD), alkali therapy for metabolic acidosis (Tab. Nodosis 500 mg TID) were started.

Patient's vitals dropped (BP-90/60 mm Hg, PR-90/min, SpO_2–76% at room air, RR-10/min) and lost consciousness within 24 hours of admission. He was maintained on 6 litres of oxygen inhalation and intermittent CPAP ventilation for few hours.

On 11-02-2018, in view of falling SpO_2 (74% at 6 litres of oxygen) and poor GCS 3/15 ($E_1V_1M_1$), patient was intubated with 8mm endotracheal tube and connected to VAC mode of ventilation.

Three days following his admission, patient had paucity of movements on left upper and lower limb. A repeat CT Brain (Fig 2) was done which showed a large intraparenchymal bleed in the right parieto-occipital region.

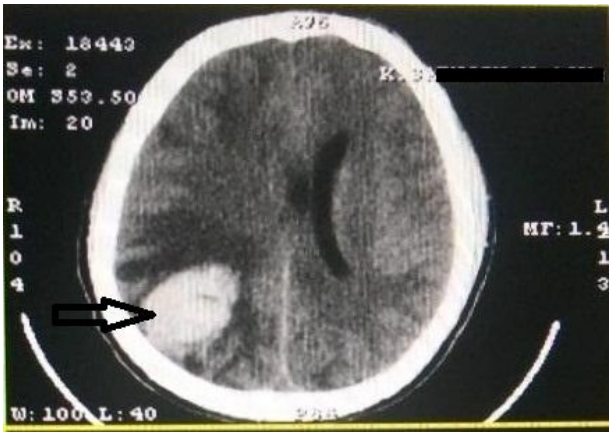

Fig 2. CTscan showing bleed in parieto-occipital region

Treatment continued with FFP, Platelet concentrate, antibiotics (as mentioned above), heparin free hemodialysis to treat acute onset of renal failure. Neurosurgeon, neurologist, nephrologist opinions were taken. Slowly patient's general condition improved, was extubated, ptosis corrected but complained of blurring of vision for which ophthalmologist opinion was taken. His condition was diagnosed as Macular edema with choroidal sclerosis for which prednisolone and timolol eyedrops were prescribed.

Renal parameters stabilised with Serum Creatinine-0.5mg/dl and patient was given prophylactic antiepileptic treatment (Tab. Phenytoin 100 mg TID). He was advised physiotherapy on OPD basis for residual (left sided) hemiparesis and discharged on 17-03-2018.

Discussion

Snake venom is a mixure made of 20 or more components. It contains protein in the form of enzyme, non enzymatic polypeptide toxins and non toxic nerve growth factors. Enzymes are digestive hydrolases, hylanurodiase, and various activators and inactivators of physiologic process. Majority of venoms consist of 1-Amino acid oxidase, phosphomono and diesterases, 5-nucleotidase, DNAse, phopsholipase A2, peptidase and NAD Nucleosides[4,5].

Viper venoms interfere with blood clotting. Russel venom is a rich source of enzymes that activate factor X to convert prothrombin to thrombin in the presence of calcium, factor V and platelets. Thus Russel venom contains several different procoagulants which activates different steps in clotting cascade. The fibrionlytic activity of viper venom is so fast that sometimes within 30 minutes of the bite the coagulation factors are so depleted that blood doesn't clot[6].

Acute bleeding is due to rapid development of DIC (Disseminated Intravascular coagulation) due to consumption coagulopathy due to conversion of procoagulant to coagulant and fibrinolysis, damage of vascular endothelium and platelet abnormalities[7].

Active bleeding is seen within 30 minutes to few hours from gums. Epistaxis occur, skin is ecchymosed, subconjunctival hemorrhages can occur. There may be hematuria, hematemesis , bleeding in peritoneal cavity, intracranial bleed and active bleeding from post partum uterus, active uncontrolled bleed from wound, abrasion or punctured site. Hence, no intramuscular injection to be given in viper bite patient as it may result in hematoma. Epigastric pain often precedes acute bleeding[8].

Respiratory paralysis due to Russell's viper is common in southern part of India and Sri Lanka. Russell's venom causes pre synaptic neuromuscular block like krait venom and resistance to anti cholinesterase. Artificial ventilator support is required for respiratory failure. Ptosis gradually occurs within 6-8 hrs and persists for one week[9].

20-40% hospitalized cases of Russell`s viper develops anuria, oliguria and acute renal failure within few hours to as late as 96 hrs due to direct nephrotoxic action of venom, heavy proteinuria, with red cell casts suggestive of glomerular damage[7].

Renal failure can be due to direct action of snake venom resulting in acute tubular necrosis, interstitial nephritis, patchy cortical necrosis and hypovolemic prerenal failure due to acute blood loss and hypotension. Early administration of ASV, mannitol and diuretic may help to delay or prevent acute renal failure[10,11].

ASV is life saving and the available anti venom in India is polyvalent. There is no need for test dose. Indications for anti venom include incoagulable blood, spontaneous bleeding from nose, urinary tract etc. Neurotoxicity like ptosis and respiratory problems; severe swelling involving half of the bitten limb or rapidly crossing a joint may occur[12].

Conclusion

In the present case, ASV therapy was started immediately on arrival to the triage and continued in view of worsening neurological status after the first dose of ASV. The PT, INR and aPTT were grossly deranged with significant drop in platelets, which mandated repeated transfusions of FFPs and packed cells. In view of decreased urine output and raised serum creatinine, patient was considered for hemodialysis which improved his renal condition. He was conservatively managed for intracerebral bleed; physiotherapy had greatly reversed his condition of hemiparesis.

Intracerebral haemorrhage is an uncommon but potentially fatal complication of viper envenomisation. Early detection with prompt initiation of ASV coupled with FFP can result in good outcome.

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Conflict of interest: None

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