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SNAKE BITE PRESENTING AS INTRACRANIAL BLEED WITH NORMAL COAGULATION PARAMETERS

Medicine		
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ABSTRACT

Snakebite being commonly encountered emergency in our country and most dreaded one too. It has been estimated that as many as 2.8 million people are bitten by snakes, and 45 900 people die from snakebite every year in India 1. The most common coagulopathy associated with snake-bite envenoming is Venom Induced Consumptive Coagulopathy. Venom contain enzymes like proteases, phospholipase A2, hyaluronidase and arginine ester hydrolase. Phospholipase A2 is the factor responsible for hemolysis secondary to the esterlytic effect on the red cell membranes The hyaluronidase causes spread of the venom in the subcutaneous tissue by disrupting mucopolysaccharides. In majority of cases there is disruption in intracerebral hemorrhage. But always it is not true there are some cases in which there is hemorrhagic risk without alteration in coagulation profile. All my 3 cases present to us with normal coagulation profile, One 26 year old male present within one hour of snake bite and died within 3 days of the bite, while other two presented lately that is 2and 6 day of the snake bite, of which both survived and had no residual focal deficit at time of discharge. This delayed clinical, laboratory manifestation of vasculotoxic snake due to the delayed seepage of venom from deeper reservoirs in the bite site or due to disassembly of the antigen-antibody complex with reinstitution of circulating unbound venom constituents. Intracranial hemorrhages are poorly understood in case of snake bite and can occur later complication also even after treatment with ASV. Still use of FFP is not advocated in much studies, there is immense need to investigate this area. Use of ASV and FFP without increased WBCT to avoid later complication is to be studied.

KEYWORDS

INTRODUCTION

- Our ancestor are being called as snake charmers and they had colossal knowledge about snakes and their bites, but still mechanism of them casing cerebral bleed is unknown and many theories are put forward to explain them. Snakebite being commonly encountered emergency in our country and most dreaded one too.
- It has been estimated that as many as 2.8 million people are bitten by snakes, and 45 900 people die from snakebite every year in India¹.
- Regardless of enough knowledge every snake is thought to be poisonous,but in reality only 15% are poisnous² Common venomous snakes include the families Elapide,Viperidae, Atractaspididae, and some of the Colubridae.
- The toxicity of venom is mainly indicated by murine Ld₅₀.

OBJECTIVE

- To study the clinical profile of the three patients who are presenting with cerebral symptoms post snake bite
- To study the possible aetiology of IC Bleed after snake bite with normal coagulation profile.

CASE 1

 A, 45 year old male farmer was admitted after 6 days of snake bite on both hands

CLINICAL FEATURES

• Pain at bite mark Agitated; Tingling sensation in left upper limb; Unable to move left upper and lower limb

NEUROLOGICAL EXAMINATION

- Conscious ,oriented; left upper and lower limb hemiparesis for past 3 days; Deviation of mouth and uvula to left
- Power 0/5 in left upper and lower limb with decreased tone in the both;Plantars ↑/-

INVESTIGATIONS

• Hb 14.7, TLC 12720,PLT 1.84 LFT NORMAL; RFT

NORMAL ELECTROLYTES NORMAL BT/CT NORMAL

• WBCT 5 min 30 sec PT/INR 12.4/1.06

Ct BRAIN

• 4.5X2.4 cm bleed in Rt high parietal lobe 2.6X 1.8 cm bleed in right occipital lobe

TREATMENT

- ASV&4 ffp given 6 days back in local hospital IV Antibiotics;Inj. Mannitol;Inj 3% Nacl infusion;Inj levetiracetam Limb physiotherapy
 Patient responsive to treatment and discharged after 2 weeks
- attent responsive to treatment and discharged after 2 weeks



CASE 2

 A, 26 year old male was bought to emergency room after hours of snake bite on right arm

CLINICALFEATURES

• Unresponsive to verbal command;Excessive bleeding from bite site and back of head(with history of trauma to head after falling from bed)

NEUROLOGICAL EXAMINATION

- Drowsy, disoriented ,not following verbal command;Right upper and lower limb tone decreased
- Power 2/5 in Rt upper limb and 0/5 in Rt lower limb; Plantars \uparrow / \uparrow

63

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INVESTIGATIONS

- Hb 13.5, TLC 14950,PLT 0.49;LFT T.Bill 2.10, SGOT 112 RFT NORMAL; ELECTROLYTESK 3.0 BT/CTNORMAL
- WBCT>20 mins PT/INR 17.8/1.52

CT BRAIN

4.4X2.3 cms ICH in left temporal lobe;3.6X1.9cms&2.2X1.2cms ICH in left frontal lobe;2.3X1.4 cms ICH in right temporal lobe 2X1.0cms in right frontal lobe;1.1X0.8 cms ICH in right high parietal lobe

TREATMENT

- ASV&4 ffp and 6 Rdp given IV Antibiotics; Inj. Mannitol Inj vitamin K;Inj tranexamic acid Inj ethamsylate;Inj levetiracetam; Limb physiotherapy
- Patient unresponsive to treatment and died within 3 days of admission



CASE 3

A, 48 year old housewife seek medical attention after 2 days of snake bite on her right middle finger

CLINICAL FEATURES

2-3 episode of vomiting after snake bite at onset;2 days later she developed sudden onset agitation and altered sensorium

NEUROLOGICAL EXAMINATION

Altered sensorium No neuro focal deficit; Power 5/5 in all 4 limbs Plantars↓/-

INVESTIGATIONS

- Hb 11.6, TLC 14500,PLT 0.93 LFT NORMAL;RFT NORMAL ELECTROLYTES (K 2.5) BT/CT NORMAL
- WBCT 4 min 20 sec PT/INR 12.5/1.06

CT BRAIN

43X39cm hematoma in Rt frontal lobe 6 mm midline shift to left

TREATMENT

ASV&4 ffp given IV Antibiotics; Inj. Mannitol; Inj 3% Nacl infusion Inj levetiracetam

Patient responsive to treatment and discharged after 2 weeks



DISCUSSION

64

The most common coagulopathy associated with snake-bite envenoming is Venom Induced Consumptive Coagulopathy.

- Thrombogenic enzymes act in conjunction with other enzymes to promote the formation of weak fibrin clots which activate plasmin and result in consumption coagulopathy and haemorrhagic consequences^[3]. Viper venom also contain prothrombin activators (ecarin and carina activase) causing variable deficiency in factor V, VII and fibrinogen [4]
- In majority of cases there is disruption in couagulation profile causing increase in PT, INR, aPTT, thrombocytopenia and increase in FDP, which suggests DIC as the probable cause for intracerebral haemorrhage ^[5]. But always it is not true there are some cases in which there is haemorrhagic risk without alteration in coagulation profile.
- There are some cases in which there is delayed clinical, laboratory manifestation of vasculotoxic snake due to the delayed seepage of venom from deeper reservoirs in the bite site or due to disassembly of the antigen-antibody complex with reinstitution of circulating unbound venom constituents. This probably explains the delayed appearance of intracerebral haemorrhage in our patient as quoted by Kularatne SAM et al., and Kumar N et al., [6.7]
- WHO guidelines dictate that 100ml of polyvalent ASV is to be given initially after skin testing, Repeat dose of 100 ml ASV is to be given one to two hours of the first dose if there is no clinical improvement or if neurological manifestations occur or the patients' condition deteriorates. ASV is continued in patients with persistence or recurrence of blood incoagulability after six hours of first dose, in patients who continue to bleed briskly after one to two hours of first dose of ASV and in deteriorating neurologic and cardiovascular signs after one to two hours of first dose of ASV.

CONCLUSION

Intracranial haemorrhages are poorly understood in case of snake bite and can occur later complication also even after treatment with ASV. Still use of FFP is not advocated in much studies, there is immense need to investigate this area. Use of ASV and FFP without increased WBCT to avoid later complication is to be studied. Mechanism of vasculotoxic snake bite causing IC bleed with normal coagulation profile is also need to be studies.

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