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General Medicine

MANAGEMENT OF SNAKE BITE IN AMCU

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ABSTRACT Snake bite is a significant health problem in India, particularly in the rural regions of the country. In general about 70% of bites are due to snakes which are not poisonous, of the rest, 15% are dry bites and only15% cause envenomation. Venom is the saliva of snake ejected during the act of biting, from the poison apparatus (the modified parotid glands). It can be, neurotoxic, vasculotoxic, or myotoxic in its action. Neurotoxicity is a key feature of some envenoming, and there are many unanswered questions regarding its manifestations. The polyvalent antisnake venom serum available in India is effective against most common poisonous snakes. Therefore, a prompt diagnosis and timely administration of polyvalent antisnake venom, in a case of snake bite can not only be life saving, but also prevent morbidity to a great extent. Neurotoxic snakes for example common krait hunt nocturnally, and are quick to bite people sleeping on the floor, often without waking their victims, since the venom is painless. Victims wake up later, paralyzed or die in their sleep. Here, we discuss the neurological manifestations, disease course and management of snake poisoning in our respiratory Intensive care unit.

KEYWORDS :

1. INTRODUCTION

Snake bite is a neglected tropical disease of global importance. [1] Data from the million deaths study in India estimates that snake bite deaths are more than 30 fold higher than recorded in official hospital returns. [2] According to toxicity, they are categorized as haemotoxic, neurotoxic, and myotoxic. Among the neurotoxic groups, the majority of bites are due to Ophiphagus Hannah (king cobra), Naja naja (common cobra), and Bungarus caeruleus (Krait) in India. There are many challenges to the study of neurotoxicity after snake bite. There is considerable variation between individual patients in the clinical manifestations following envenoming by any particular species. Clinical manifestations are related to hypotension, shock and other organ dysfunction (such as renal impairment). manifestations of envenoming by the non-neurotoxic are such as those due to coagulopathy. Comparing findings from different studies is difficult as there is a lack of uniformity in description or grading of neuromuscular weakness, or in assessment of response to treatment. Interpretation of neurophysiological findings is also difficult as different methods have been used between studies. [3] However, timely administered antisnake venom and ventillatory assistance can prevent the mortality and morbidity of the victims. Snakebite poisoning is one of the common causes of admission in intensive care unit. Two cases are being reported to show the variance in clinical presentation and outline management protocols in Respiratory intensive care unit.

CASE SCENARIO 1:

A female patient name yyy, aged 38 years, with Ip no.19910 resident of Gudlavalleru, KRISHNA DIST, came to hospital with alleged history of snake bite on right ring finger on 23/09/2018 at 4 am at her residence. She developed giddiness and breathlessness after the bite which is increased in severity and became slowly unconscious at around 9 am. She was taken to a private hospital and was given Inj ASV 10 vials and inj.TT 0.5 mg IM and then Intubated there and shifted to GGH Vijayawada. At our hospital o/E patient is unconscious with GCS of E1Vet M1, VITALS BP 120/80mmhg, PR 104/min, afebrile,no petechiae,no hematuria and bleeding gums.CVS examination reveals normal heart sounds**, Respiratory system** examination shows B/l air entry equal with ET insitu and no added sounds. Patient was treated with AntiSnakeVenom 5 vials, inj.atropine 0.6 mg IV stat + inj.neostigmine 1.5mg i.v stat. The same repeated every 30 min. up to 5 doses along with inj.ceftrioxone 1gm iv, inj.pantop 40mg, inj. Metrogyl 100mg iv.No improvement is observed with this treatment in this patient.

With the history of early morning bite while sleeping on floor krait bite was suspected and given inj.calcium gluconate 1gm iv stat then there was improvement in motor power.Inj calcium gluconate 1gm iv b.d continued for 7 days. Patient slowly weaned off from ventilator and extubated after 3days and discharged after 4 days.Patient general condition and vitals were stable at the time of discharge.

2nd Case report : AMCU management of a case of snake bite.

A 28 year old male patient presented to the causality alleged to have sustained snake bite over dorsum of hand. Patient was under the influence of alcohol. There was history of vomiting, Altered sensorium , bleeding from bite site and drooping of eyelids .there was history of decreased urine output, Hematuria, Bleeding gums, Unable to lift head above the pillow. The past medical history and family history was not significant. On examination patient was in altered sensorium. There was no pallor, icterus, cyanosis, clubbing, lymphaedenopathy, pedal edema. Vitals: BP : 90/60 mm Hg in supine position . PR: 80bpm , regular in rhythm, low volume pulse. Temp: 98.4F SpO2: 97 % in room air.

Systemic examination: CNS : Patient in altered sensorium , ptosis , Neck muscle weakness. The power of right upper limb was 2/6 ,left upper limb 2/6, right lower limb 2/6 and left lower limb 2/6.

Examination of other systems was normal.

INVESTIGATIONS:

Whole blood clotting time at the time of admission was 69 minutes. Thrombocytopenia (38000/cumm) .PT: 42sec , INR: 3.08, APTT: 84 sec. Hb : 5.5 gm/dl , elevated reticulocyte count 3%.Indirect bilirubin: 3.5 mg/dl, Total bilirubin: 4.5 mg/dl. Raised lactate dehydrogenase: 300mg/dl all suggestive of hemolytic anemia. Serum Creatinine: 4.1mg/dl.

TREATMENT:

Inj. Atropine 1 mg IV stat, inj. Neostigmine 2mg im stat.
Inj. Atropine 1 mg IV drip started Inj. Neostigmine 0.5 mg iv every 30min.
Inj ASV 20 vials in 1unit NS.
4 units FFP given, Ryles tube inserted, Antibiotics started.
Patient was Intubated in view of low GCS and desaturation with 8 ID ETT & connected to a mechanical ventilator.

Mode: VCV TV : 480 ml. RR : 16 BPM PEEP : 4 cm H20 Fio2 : 1 ----- --- 0.4 over 4 hours.

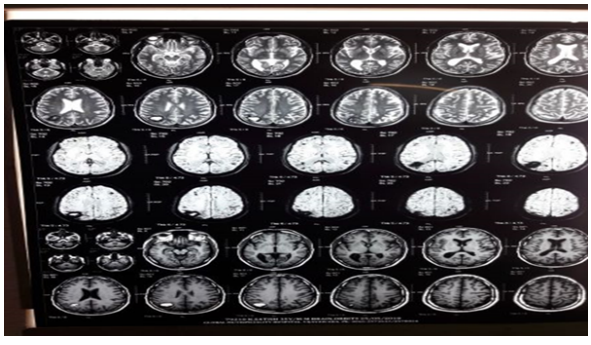
Patient developed Right upper limb cellulitis, hemiparesis Glycerine & MgSO4 dressing was done. With Limb elevation Tab,. Serratiopept idase 10mg TID started

DAY 2 :
Black colored urine. 50ml in 24 hrs
Total 60 vials of ASV. Given 4 units FFPs given.
BP ; 190/110 mm Hg , supine position
Inj . Frusemide 40mg BD started.

DAY 3:
Se. creatinine : 6.6
Heparin free hemodialysis started.
BP : 210/ 120 mm Hg
Inj . Furosemide 40 mg TID, T.Clonidine 0.1 mg TID, T.Clindipine 20Mg BD Started.

DAY 6:
Patient developed ARDS . PaO2; 110 With Fio2 of 1. PEEP : 8 Cm of H2O.
Chest x-ray revealed diffuse infiltrates, suggestive of ARDS.

DAY 7:
A repeat CT brain was done which showed a large intra parenchymal bleed in the right parieto occipital region.

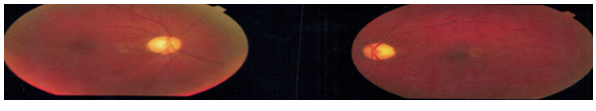


DAY 10:
Coagulation profile : normal RT feeds started

DAY 12 :
Percutaneous tracheostomy was done. 7.5 ID Tracheostomy tube was inserted
Connected to mechanical ventilator.

DAY 16 :
AKI Resolved. Se.Creatinine 1.0 Urine output improved.
Patient become conscious & is obeying commands.Weaning started.
Patient on PSV.

DAY 18 :
Ptosis corrected. Patient on T- piece
Patient complained of blurring of vision .
His condition was diagnosed as Macular edema with choroidal sclerosis for which Prednisolone and Timolol eye drops were prescribed initially.



Slowly the vision loss progressed to be diagnosed as Bilateral Optic Atrophy within 2 months after discharge.

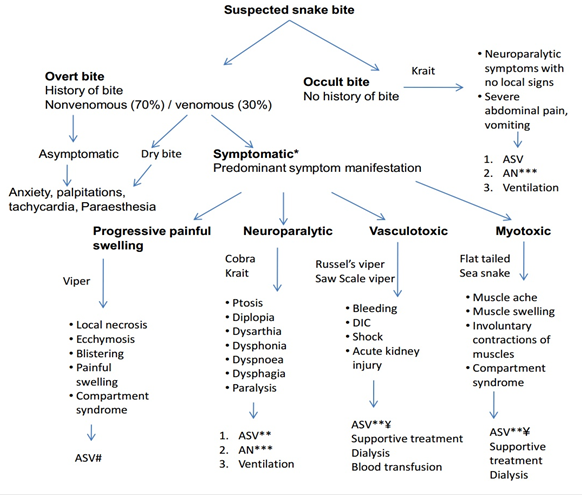
His vision had improved from perception of light to counting of fingers at a distance of one feet with intravenous steroid therapy for few days.
His vision now is 6/60

His hemiparesis was completely resolved with regular physiotherapy

III.DISCUSSION

Snake venom contains several types of polypeptide toxins, of which the neurotoxins produce paralytic effect by binding to presynaptic and post synaptic sites at neuromuscular junction. [4] Common neurological symptoms in decreasing order of frequency include ptosis (85.7%), ophthalmoplegia (75%), limb weakness (26.8%), respiratory failure (17.9%), palatal weakness (10.7%) and neck muscle weakness (7.1%). These are experienced usually within 6 hours of bite. (5) Following administration of antivenom, the signs of recovery become evident written a few hours to several days. [6. Prompt recognition of envenomation and timely administration of antisnake venom (anti-sera) is a life saving measure and is the only effective treatment for neutralization of toxins that has entered the circulation. [7] Polyvalent antivenom has no significant benefit in reversing respiratory paralysis and preventing delayed neurological complications. Polyvalent ASV is relatively safe, and allergic reactions after ASV injection can be prevented by premedication with adrenaline, IV hydrocortisone and antihistaminic. [8] Anticholinest erases are beneficial against the postsynaptic toxins that induce myasthenia like block. [9] In animal models, subjected to high dose of snake venom, anticholinesterases have proven their efficacy as antidote in extending expected survival time. [10] However, despite their proven efficacy anticholinesterase, and antisnake venom forms mainstay of therapy, and dose up to 400 ml have been used. In present case, 14 vials of polyvalent antisnake venom were used. Ventillatory support forms a cornerstone of envenomation therapy. Incidence of complication is directly proportional to the duration of venom in blood. Respiratory failure is the most common cause of mortality and morbidity in victims bitten by snakes. A mortality rate of 7.6% was observed in patients on intensive care management. A prompt recognition of respiratory failure and timely mechanical ventilation can decrease morbidity and mortality .But due to poor availability at periphery and at larger district centre ASV, still remains mainstay of therapy. [8]

Krait poison containing Beta Bungaro toxin affects presynaptic fibres where calcium ion act as neuro transmitter. If there is no improvement with 3 doses of Atropine+ Neostigmine , Inj 10% calcium Gluconate10 ml slow iv over 10 minutes every 8-12 hours for 5-7 days until recovery from neuroparalysis is advised as per literature



* Even though present as predominant manifestation but there may be overlap of syndrome as well.
\# ASV indicated in rapidly developing swelling only. Purely localized swelling with or without bite marks is not an indication of ASV.
** For reaction to antisnake venom (ASV) Dose of Adrenaline 0.5 mg IM (in children 0.01 mg/kg)
¥ Specific ASV for sea snake and Pit viper bite is not available in India. However, available ASV may have some advantage by cross reaction.
*** Atropine 0.6 mg followed by neostigmine (1.5mg) to be given IV stat (In children Inj. Atropine 0.05 mg/kg followed by Inj. Neostigmine 0.04 mg/kg IV.) Repeat neostigmine dose 0.5 mg (in children 0.01mg/kg) with atropine every 30 minutes for 5 doses. Thereafter taper dose at 1 hour, 2 hour, 6 hours and 12 hour. Positive response is measured as 50% or more recovery of the ptosis in one hour. If no response after 3rd dose. Stop AN injection.

Figure 1. Four presenting clinical syndromes of snakebite i.e. progressive weakness (neuroparalytic/neurotoxic), bleeding (vasculotoxic/haemotoxic), myotoxic and painful progressive Swelling and its management.

1V.CONCLUSION

AMCU management of snake poisoning requires continuous

monitoring of clinical signs and symptoms and active intervention for positive outcome.

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