MANAGEMENT OF SNAKE BITE

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INTRODUCTION

• Snakes are curious creatures among all animals known to man.
• They are intrinsically fascinating, awe inspiring.
• We have countless superstitions about them.
• In the race of evolution snakes become more successful & specialized due to their elongated body
• …slithering serpentine movement
• …absence of legs, eyelids, ears (chakshushraba) & even vocal cords to produce true voice.
INTRODUCTION contd.

- The world population of snake is comprised of about 2800 species of which only 375 species are known to be poisonous.
- India has always had the somewhat unenviable reputation of harboring some of the world’s most venomous snakes.
- India has 244 species of which only 57 species are poisonous & deadliest are Cobra, Krait & Viper.
INTRODUCTION contd.

• Snake bite per anum roughly 200,000 of which 20,000 prove fatal.

• Snake everywhere except in Arctic lands, Newzeland & Ireland.

• Australia where 90% snakes are venomous, has the species with most toxic venom on earth.
CLASSIFICATION OF POISONOUS SNAKE

• Elapidae:- Common Cobra (Gokhura, Kaouthia—1) Monocled- Naja naja Kaouthia. 2) Diocellate N n naja. 3) Aocellate N n oxiana.
• ....King Cobra
• ...........:- Krait—1) Common Krait-Chitti. 2) Banded Krait – Sankhini.
• Viperidae--(1) Pitless Viper- (a)Russell’s Viper – chandrabora. (b) saw-scaled viper-echis carinata(body scale has serrated edge).
• ............... (2) pit viper(crotalidae).
• Sea snake
A Canalised fang of a Russell's Viper to the left; Grooved fang of a Cobra to the right.

Head of a Russell's Viper showing the poison glands and the cannulated fangs. A—Fangs, B—Curved teeth. C—Tongue, D—Orifice of the fang, E—Sheath over the fang, F—Poison duct at base of the fang, G—Poison gland.

(From P. J. Decoras—SNAKES OF INDIA)
Scales on the head of a Russell’s Viper. Note triangular head with small scales.

Ventral scales in a poisonous snake extending right across belly.

Ventral scales in a non-poisonous land snake. They do not extend right across the belly.
Differences between poisonous & non-poisonous snakes:

<table>
<thead>
<tr>
<th></th>
<th>Poisonous</th>
<th>Non-poisonous</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>Belly scale</td>
<td></td>
</tr>
<tr>
<td></td>
<td>Large – cover the entire breath of body</td>
<td>Small, like those on the back, will never cover the entire breath of belly.</td>
</tr>
<tr>
<td>2</td>
<td>Head scale</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Usually small in vipers</td>
<td>Usually large</td>
</tr>
<tr>
<td></td>
<td>• May be large, in pit viper (but they have pit between eyes &amp; nostrils)</td>
<td></td>
</tr>
<tr>
<td>3</td>
<td>Fangs</td>
<td></td>
</tr>
<tr>
<td></td>
<td>• Hollow like hypodermic needle</td>
<td>Short &amp; solid</td>
</tr>
<tr>
<td></td>
<td>Poisonous</td>
<td>Non-poisonous</td>
</tr>
<tr>
<td>---</td>
<td>-----------------------------------------------</td>
<td>----------------------------------------------</td>
</tr>
<tr>
<td>4. Tail</td>
<td>Compressed</td>
<td>Non markedly compressed</td>
</tr>
<tr>
<td>5. Habits</td>
<td>Usually nocturnal</td>
<td>Not so</td>
</tr>
<tr>
<td>6. Teeth-bite marks</td>
<td>Two fang marks are left at the site of bite usually with or without marks of other teeth</td>
<td>In addition to 2 fang marks, there will be a number of small teeth marks on these snakes have several small teeth.</td>
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</tbody>
</table>
Management of Snake Bite

FIRST AID

1. Re-assure
2. Immobilize the bitten limb using a splint or sling. If available firm binding of the splint with a crepe bandage is an effective form of immobilization.
3. Muscular contraction in the bitten limb will promote spread of venom.
Rejected controversial first aid methods

1. Cauterization  
2. Incision or excision  
3. Amputation of digits  
4. Suction by mouth  
5. Vacuum pumps  
6. Instillation of chemical compounds like KMnO4  
7. Cooling with ice  
8. Electric shock  

None of these methods aimed at removing venom from the site of the bite has received consistent support from the result of animal experiment.

The use of tourniquets, compression pads and bandages in an attempt to impede the systemic spread of venom, remains controversial.
The splint & crepe bandage is certainly an effective way of immobilizing the bitten limb, and is a less painful way of applying obstructive pressure than is the arterial tourniquet. However, in practice, it is difficult to judge how tightly to apply the crepe bandage, difficult for the patient to apply unaided & might accentuate the locally necrotic effects of some snake venoms.

Dangers of tourniquet & other occlusive methods include ischemia & gangrene, if they are applied for more than about 2 hours, damage to peripheral nerves, increased fibrinolytic activity, congestion, swelling, increased bleeding, increased local effect of venom & shock or rapid development of life-threatening systemic envenoming after their release.

Tourniquet/constricting band should be strongly discouraged.
However if a patient is bitten by a dangerous neurotoxic elapid & medical attention is likely to be delayed for 1-2 hours, & there is a risk that respiratory paralysis might develop en route to hospital. In these cases alone it seems reasonable to apply a firm crepe bandage & splint (if available), or a tight tourniquet (upper arm or thigh) in the hope of delaying the onset of life-threatening neurotoxicity until the patient reach a place where they can be resuscitated.
TREATMENT OF EARLY SYMPTOMS

• *En route
• For Local pain – oral paracetamol-not aspirin
• Severe pain– pethidine or pentazocine
• Vomiting – I.V. chlorpromazine
• Syncopal attack – VVA etc.– head end down etc.
• Allergic manifestation--Chlorpheniramine maleate
• Hypotension or bronchoconstriction – treated with adrenaline 0.1% (1 in 1000)( 0.5ml) by s/c inj.
TREATMENT AT HOSPITAL

• *Clinical assessment
• 3 most important preliminary questions
• 1—Which part of body bitten
• 2—How long ago bitten
• 3—Have you brought the snake or, if not, did you see what kind of snake it was?
• Fang marks – sometime invisible & rarely help in diagnosis, although 2 or 3 discrete puncture marks suggests fangs of venomous snake.
• Local swelling /skin lesion / bleeding from puncture site?
• Shock – sweating, cold clammy extremities, collapsed pulse, low B.P., tachycardia – Rx I.V. fluids, plasma expanders – FFP, Dextran, Hemacel or Fresh blood.
* Early symptoms of Neurotoxicity after Elapid bites – blurred vision, feeling of heaviness in the eyelids & drowsiness / earliest sign – contraction of frontalis muscle (raised eyebrows & puckered forehead) even before true ptosis can be demonstrated.

Signs of respiratory muscle paralysis (dyspnoea, exaggerated abdominal respiration & cyanosis) must be detected.

Dark urine suggests haemoglobinuria or myoglobinuria. In absence of the above symptoms – the pt. kept admitted for at least 24 hours & watched for the above symptoms every hourly.

Useful Investigations– whole blood clotting time, P-time, PTT, PBC, CPK, SGOT, Urea, Creatinine, Electrolytes. Urine - albumin, hemoglobin, myoglobin & microscopy – RBC etc.
TREATMENT AT HOSPITAL

• A S V (Anti Snake Venom) is the whole serum or enzyme-refined immunoglobulin of animals, usually horses or sheep, which has been immunised with snake-venom.

• It is the only specific treatment. In the management of snake bite, the most important clinical decision is whether or not to give antivenom, for only a minority of snake-bitten patients need it.

• It may produce severe reaction & it is expensive & often in short supply.
INDICATION FOR ASV

• *SYSTEMIC ENVENOMING:
  • 1) Haemostatic abnormalities:
  • spontaneous systemic bleeding, incoagluable blood or prolonged CT, elevated FDP, thrombocytopenia.
  • 2) Cardiovascular abnormalities:
  • hypotension, shock, abnormal ECG, cardiac arrhythmia, cardiac failure, pulmonary edema.
  • 3) Neurotoxicity
4) Generalized rhabdomyolysis

5) Impaired consciousness

7) In patients with definite signs of local envenomining, the following indicate significant systemic envenomining: neutrophil leucocytosis, elevated serum enzymes such as CPK & aminotransferases, haemoconcentration, uremia, hypercreatininaemia, oliguria, hypoxaemia, acidosis & vomiting.

*SEVERE LOCAL ENVENOMING with rapid spread, even in absence of systemic envenomining*
The major limitation to successful use of ASV has been practice of empiric dosage, the reason for this is obvious:

1) Most of the time the snake is not identified.
2) Symptomatology of the patient may not be an accurate guide to decide the dose of ASV.
3) Accurate titration of dose can only be possible if venom antigen level in the blood can be repeatedly measured till its total disappearance from body.
4) Potency of ASV may be occasionally lost. However, this can be easily identified when lyophilized polyvalent ASV after reconstitution with diluents appear opaque (instead of clear) which suggests precipitation of protein & thus reduced potency.

Presently available lyophilized ASV retains its potency for at least 5 years & does not require storing in refrigerator (storing in cool dark place is adequate).
CONSIDERATION BEFORE STARTING ASV

1) Identification of poisonous snake bite

2) Dry bite – bite without venom being injected – watch for 24–48 hrs, if no signs of envenomation – no ASV.

3) Local infiltration of ASV – not beneficial.

4) Hypersensitivity to horse antiserum: always a potential risk – so be sure envenomation has occurred. Prior intradermal or s/c test has no predictive value for early (anaphylactic) or late (serum sickness type) reactions.

Do not waste time if ASV is indicated.

If h/o atopy, pretreatment with adrenaline, antihistaminic & corticosteroids.
Each ml. of reconstituted polyvalent ASV serum available in India neutralizes not less than the following quantities of standard venom (when tested in white mice):-

...Cobra- 0.6mg, Russell’s Viper- 0.6mg,
...Common Krait- 0.45mg, Saw-scaled Viper- 0.45mg.
• Pathological effect of viper venom (vv) may not be noticed until about 6 hrs of bite (vary between 1.5-72 hrs).

• The vv may remain functionally active causing persistent coagulopathy even after 3 weeks of bite.

  > This is probably the result of continuing absorption of venom from bite site.

  > ASV clears the venom from circulation immediately, the clinical effects on clotting restoration occur usually after 4 hrs – so repeating CT too soon after initial dose of ASV, may be misleading.
Half-life of ASV is only 26-95 hrs whereas antigen may reappear in circulation as long as 130 hrs after initial correction of coagulopathy due to contd. absorption of venom from bite site, which acts as a depot & slowly release venom.

Coagulation defect due to venom may be transiently reversed by use of fresh blood / FFP, however it is likely to recur. Thus blood transfusion can be used only as an adjunct to ASV, to correct blood loss & resulting shock.
EARLY USE OF ASV JUSTIFIED?

Reid in 1968 suggested use of ASV only when pt. starts bleeding. (CT ignored?)

> However because of risk of SAH, abortion, irreversible shock, ARF etc., such practice is abandoned & now any pt. showing prolonged CT irrespective of clinical bleeding, is immediately administered ASV.
NEVER TO LATE

Misconception – ASV is unlikely to work after 2-3 days of bite.

But functionally active venom (neutralisable by ASV) is present in circulation even 3 weeks after bite.

So ASV is likely to work even when started very late due to late reporting of patient.
Most ideal way—titrating ASV dose against venom antigen level (measured by ELISA) – not practicable.

Bed-side study of coagulation profile, as it correlates very well with blood venom level. They are (1) Blood Clotting time (Lee & White method) & (2) Clot quality test.

Systemic envenomation is graded as:

<table>
<thead>
<tr>
<th>Envenomation</th>
<th>CT</th>
<th>Clot Size</th>
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<tbody>
<tr>
<td>Mild</td>
<td>15’</td>
<td>&lt; 50 % of whole blood</td>
</tr>
<tr>
<td>Moderate</td>
<td>15’-30’</td>
<td>A small speck</td>
</tr>
<tr>
<td>Severe</td>
<td>Incoagulable blood</td>
<td></td>
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</tbody>
</table>

Occasionally normal CT, but clot quality is poor, not firm — considered as coagulation defect – ASV to be given.
MODE OF ADMINISTRATION OF ASV IN VIPER BITE

• 3 MODES:

• 1) Single large bolus i.v.– ABANDONED, since ASV has limited half life & thus is not capable of preventing late envenomation.

• 2) Intermittent (small) bolus doses: (MOST POPULAR) ... Intermittent i.v. bolus doses of ASV, usually every 6hrs until signs of systemic envenomation disappear.

...ASV restores normal CT. after 4hrs.

...Advantage:- no misuse of larger dose, better control of coagulation & late envenomation
3) Continuous I V infusion with intermittent bolus :-

Adv:- This may be the best method for exactly titrating the dose of ASV. It is likely to reduce the total dose of ASV as compared even to previous method .

Disadv:- Slower correction of preventable life – threatening bleeding may prove costly .
RELAPSE OF BLEEDING

In a fair no. of cases relapse may occur as long as 130hrs after initial correction.
## Dosage Regimen of AVS

<table>
<thead>
<tr>
<th>Clinical spectrum</th>
<th>Dosage of AVS</th>
</tr>
</thead>
<tbody>
<tr>
<td>1. Dry bite- no sign of envenomation</td>
<td>No AVS</td>
</tr>
<tr>
<td>2. Bites with local swelling without any systemic features</td>
<td>20-50ml</td>
</tr>
<tr>
<td>3. Swelling beyond bitten site &amp; mild systemic features or bleeding</td>
<td>50-100ml</td>
</tr>
<tr>
<td>4. Marked local &amp; systemic features with hemolysis &amp; clotting abnormalities</td>
<td>100-200ml</td>
</tr>
</tbody>
</table>
Procedure of administration

• Appropriate dose in 500ml of NS & infusion rate 15-20 drops/min & progressively increasing the rate so as to complete by 1-2 hrs. If no improvement supplemental doses may have to be repeated. Inj. Hydrocortisone & Inj. Pheneramine maleate should be given prior to AVS.

• Prior to start AVS Infusion a 1:10 NS diluted test dose of AVS in a healthy site with a saline control on a second site. Observe every 15 mins. Positive if there is erythema. Keep inj. Adrenaline (.5ml of 1:1000 dil) & inj. Hydrocortisone (200mg) ready to combat anaphylaxis & start the desensitizing dose.
# Densensitising dose of AVS

<table>
<thead>
<tr>
<th>Time (min)</th>
<th>AVS Dose</th>
<th>If no reaction</th>
<th>If reaction is present</th>
</tr>
</thead>
<tbody>
<tr>
<td>15</td>
<td>0.1ml IM</td>
<td>Go to next dose</td>
<td>Give adrenaline S/C &amp; hydrocortisone 200mg IV</td>
</tr>
<tr>
<td>30</td>
<td>0.5ml</td>
<td>Do</td>
<td>Do</td>
</tr>
<tr>
<td>45</td>
<td>1.0ml</td>
<td>Do</td>
<td>Do</td>
</tr>
<tr>
<td>60</td>
<td>0.5ml</td>
<td>Do</td>
<td>Do</td>
</tr>
</tbody>
</table>
Venom of Cobra & Krait is mainly neurotoxic:

- Indian Cobra (Naja naja)/ Cobrotoxin/ Causes postsynaptic blockage i.e., bind to ACH receptor on motor end-plate & prevent depolarizing action of ACH.

- Common Krait (Bangerus caeruleus)/
  1. $\alpha$ bungerotoxin – action as above./
  2. $\beta$ bungerotoxin—causes presynaptic blockage i.e., prevents release of ACH at neuromuscular junction.
IDENTIFICATION OF ENVENOMATION

• Local envenomation may be prominent in cobra bite only with severe pain, swelling, tissue necrosis & blister at site of bite.

• Systemic envenomation following both cobra & krait bite (may appear between 15 minutes to 10 hrs of bite):-

  …Early features are blurred vision, ptosis or drowsiness.
  Late features:- respiratory & glossopharyngeal paralysis.
DOSAGE SCHEDULE IN ELAPIDAE BITE

• Initial dose:- 200ml preferred, 100ml may be effective. Larger dose may be required for larger snake.

• Repeat dose:- 50-100ml every 4-6hrs until neurotoxic signs disappear.

• King Cobra:- 1000ml of monospecific ASV.
• Neurotoxic signs may appear within 30mins

• Neurotoxicity may also be acutely reversible with anticholinesterase in absence of ASV.

• Mechanical ventilation alone may suffice for respiratory paralysis in absence of ASV.

• Combination of above measures may reduce total requirement of ASV.
Antivenom Reactions – 3 types

- Early anaphylactic type: Reactions usually develop within 10 to 180 minutes of starting antivenom. There is itching, urticaria, fever, tachycardia, palpitations, cough.

- The reported incidence varies from 3 to 54%, up to 40% of patients with early reactions show feature of severe systemic anaphylaxis.
Early reactions respond readily to adrenaline S/C and antihistamine.

Pyrogenic reactions – result from contamination of the antivenom by endotoxin like compounds.

High fever with rigor followed by hypotension.
Late reactions
(Serum sickness type)

- Develop 5-24 days after treatment. The higher the dose of antivenom the higher the incidence of those reactions.
- Symptoms include fever, itching, urticaria, arthralgia, mononuritis multiplex rarely encephalopahy.
SUPPORTIVE TREATMENT

- Respiratory failure.
- Hypotension & shock.
- Renal failure.
- Local infection.
- Intracompartmental syndrome.
- Snake venom ophthalmia.
- Heparin, antifibrinolytic agents such as aprotinin & EACA, trypsin & a variety of herbal remedies have been used – most are potentially harmful & none has been proved to be effective.
Mechanism of ARF

a) Hypotension: Due to vasodilatation by autacoids released by Snake venom; which are ACE inhibitor

b) Intravascular Haemolysis: Russell’s Viper
a) Disseminated intravascular coagulation:

* colubrid venoms activate complement via alternative pathway,

* Russell’s viper via classical pathway

→ Consumption coagulopathy

d) Bleeding –

(1) Vessel wall damage by Haemorrhagins

(2) Thrombocytopenia
e) Direct toxic effect of venom on the renal tubules

f) Rhabdomyolysis and Myoglobinuria by sea snake bite
PREVENTION

• Protective clothing – boots, socks, long trousers.
• Carrying light at night.
• Particular care during collecting firewood, moving logs, debris likely to conceal a snake.
• Wadding in sea.
• Rodent-proofing for domestic animals, chickens etc.
• Use of toxic chemicals – lethal or repellant to snakes.
PROPHYLACTIC IMMUNISATION

• Venom toxoids (venoids) – to immunise farmers at high risk of snake bite. Some protection against organ damage achieved.

• The production & modification of venom antigens by genetic engineering is an exciting new development which could lead to the production of snake venom vaccine.
CONCLUSION

• Since accurate measurement of venom antigen level in blood is not possible, dose of ASV used to neutralize venom has been empirical in snake bite envenomation.

• Ensuring definite envenomation prior to starting ASV is paramount, since dry bites are not uncommon.
• Multiple smaller doses of ASV every 4-6 hours are likely to be more effective in controlling bleeding in Viperine bite with hemotoxicity than a single large dose.

• Relapse of bleeding after initial correction is a distinct possibility and thus patient should be observed to give repeat dose of ASV when required.
• Elapidae bite with neurotoxicity should be promptly treated with ASV.

• Problem of shortage of ASV with regard to treating neurotoxicity, if faced, might be overcome by offering other effective supportive treatment, e.g., administration of neostigmine injection and placing patient on mechanical ventilation, till effect of neurotoxin wares off in due course of time.