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Envenomation -1

Snake Bite

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Introduction

Ophitoxaemia is the rather exotic term that characterizes the clinical spectrum of snake bite envenomation. Of the 2500-3000 species of snakes distributed world-wide, about 500 are venomous. The major families in the Indian subcontinent are: Elapidae which includes common cobra, king cobra and krait, Viperidae which includes Russell's viper, pit viper and saw-scaled viper and Hydrophidae (the sea snakes). Of the 52 poisonous species in India, majority of bites and consequent mortality is attributable to 5 species viz. Ophiophagus hannah (king cobra), Naja Naja (common cobra), Daboia rusellii (Russell's viper), Bungarus caeruleus (krait) and Echis carinatae (saw-scaled viper). In India 35,000-50,000 lives are lost per year due to venomous snake bite. More than 2000 deaths per year are reported from Maharashtra. This is the tip of the iceberg as the majority of snake bite deaths go unreported as many villagers go to traditional healers like mantriks and tantriks.

Venom composition

More than 90% of snake venom (dry weight) is protein. Each venom contains more than a hundred different proteins: enzymes (constituting 80-90% of viperid and 25-70% of elapid venoms), non-enzymatic polypeptide toxins, and non-toxic proteins.

Venom enzymes

Snake venom, the most complex of all poisons is a mixture of enzymatic and non-enzymatic compounds as well as other non-toxic proteins including carbohydrates and metals. There are over 20 different enzymes including phospholipases A2, B, C, D hydrolases, phosphatases (acid as well as alkaline), proteases, esterases, acetylcholinesterase, transaminase, hyaluronidase, phosphodiesterase, nucleotidase and ATPase and nucleosidases (DNA & RNA). The non-enzymatic components are loosely categorized as neurotoxins and haemorrhagens. Different species have differing proportions of most, if not all of the above mixtures- this is why poisonous species were formerly classified exclusively as neurotoxic, haemotoxic or myotoxic. The pathophysiologic basis for morbidity and mortality is the disruption of normal cellular functions by these enzymes and toxins. Some enzymes such as hyaluronidase disseminate venom by breaking down tissue barriers. The variation of venom composition from species to species explains the clinical diversity of ophitoxaemia. There is also considerable variation in the relative proportions of different venom constituents within a single species throughout its geographical distribution, at different seasons of the year and as a result of ageing.

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Table 1: Snakes with venom yield, lethal dose, antivenin neutralizing dose.

Snakes	Venom yeild per bite	Lethal dose for man	1ml of antivenom neutralised
Cobra	0.2 gram	0.12 grams	0.6mg
Krait	0.022 gram	0.06 grams	0.45 mg
Russell's viper	0.15 gram	0.15 grams	0.6 mg
Echis carnatus	0.0046 gram	0.08 grams	0.45 mg

Quantity of venom injected at a bite, "dry bites"

This is very variable, depending on the species and size of the snake, the mechanical efficiency of the bite, whether one or two fangs penetrated the skin and whether there were repeated strikes. Either because of mechanical inefficiency or the snake's control of venom discharge, a proportion of bites by venomous snakes does not result in the injection of sufficient venom to cause clinical effects. About 50% of bites by Russell's vipers, 30% of bites by cobras and 5%-10% of bites by saw-scaled vipers do not result in any symptoms or signs of envenoming

Although large snakes tend to inject more venom than smaller specimens of the same species, the venom of smaller, younger vipers may be richer in some dangerous components, such as those affecting haemostasis.

Recommendation: Bites by small snakes should not be ignored or dismissed. They should be taken just as seriously as bites by large snakes of the same species.

Symptoms and signs of snake-bite

When venom has not been injected

Some people who are bitten by snakes or suspect or imagine that they have been bitten, may develop quite striking symptoms and signs even when no venom has been injected. Vasovagal, collapse with slowing of heart rate, might over breathe and develop tetany.

When venom has been injected

Local symptoms and signs in the bitten part:

Fang marks, local pain, local bleeding bruising lymphangitis, lymph node enlargement inflammation, blistering local infection, abscess formation necrosis.

Generalized (systemic) symptoms and signs:

General

Nausea, vomiting, malaise, abdominal pain, weakness, drowsiness, prostration.

Cardiovascular (Viperidae)

Visual disturbances, dizziness, faintness, collapse, shock, hypotension, cardiac arrhythmias, pulmonary oedema, conjunctival oedema (chemosis)

Bleeding and clotting disorders (Viperidae)

Traumatic bleeding from recent wounds (including prolonged bleeding from the fang marks and from old partly-healed wounds Spontaneous systemic bleeding - from gums, epistaxis, bleeding into the tears, intracranial haemorrhage (meningism from subarachnoid haemorrhage, lateralizing signs and/or coma from cerebral haemorrhage, haemoptysis, haematemesis, rectal bleeding or melaena, haematuria, vaginal bleeding, bleeding into the mucosae, skin (petechiae, purpura, discoid haemorrhages and ecchymoses) and retina.

Neurological (Elapidae, Russell's viper)

Drowsiness, paraesthesiae, abnormalities of taste and smell, "heavy" eyelids, ptosis, external ophthalmoplegia, paralysis of facial muscles and other muscles innervated by the cranial nerves, nasal voice or aphonia, regurgitation through the nose, difficulty in swallowing secretions, respiratory and generalized flaccid paralysis.

Skeletal muscle breakdown (sea snakes, some krait species)

Generalized pain, stiffness and tenderness of muscles, trismus, myoglobinuria, hyperkalaemia, cardiac arrest, acute renal failure.

Renal (Viperidae, sea snakes)

Loin pain, haematuria, haemoglobinuria, myoglobinuria, oliguria/anuria, symptoms and signs of uraemia.

Endocrine (acute pituitary/adrenal insufficiency from infarction of the anterior pituitary)

Acute phase: Shock, hypoglycaemia Chronic phase (months to years after the bite): Weakness, loss of secondary sexual hair, loss of libido, amenorrhoea, testicular atrophy, hypothyroidism etc

Locked in syndrome - Few cases develop quadriplegia with total opthalmoplegia and dilated pupils. The clinician may feel the patient is brain dead or comatose, but such victims recover totally within 3-4 days if treated properly by maintaining oxygen saturation with proper ventilator support and electrolytic balance and nutrition and care of infection. This phenomenon is due to blocked postsynaptic acetyl choline receptors including the sphincter pupillary muscle which are rich in acetyl choline receptors.

Long-term complications (sequelae) of snake-bite

At the site of the bite, loss of tissue may result from sloughing or surgical débridement of necrotic areas or amputation: chronic ulceration, infection, osteomyelitis, contractures, arthrodesis or arthritis may persist causing severe physical disability. Malignant transformation may occur in skin ulcers after a number of years

Symptoms and signs of cobra-spit ophthalmia (eye injuries from spitting cobras)

If the "spat" venom enters the eyes, there is

immediate and persistent intense burning, stinging pain, followed by profuse watering of the eyes with production of whitish discharge, congested conjunctivae, spasm and swelling of the eyelids, photophobia, clouding of vision and temporary blindness. Corneal ulceration, permanent corneal scarring and secondary endophthalmitis are recognised complications of African spitting cobra venom but have not been described in Asia.

First Aid Treatment Protocol

Much of the first aid currently carried out is ineffective and dangerous.

Recommended Method for India

The first aid being currently recommended is based around the mnemonic:

"Do it R.I.G.H.T."

It consists of the following:

R. = Reassure the patient. 70% of all snakebites are from non- venomous species. Only 50% of bites by venomous species actually envenomate the patient

I = Immobilise in the same way as a fractured limb. Use bandages or cloth to hold the splints, not to block the blood supply or apply pressure. Do not apply any compression in the form of tight ligatures, they don't work and can be dangerous!

G. H. = Get to Hospital Immediately. Traditional remedies have NO PROVEN benefit in treating snakebite.

T= Tell the doctor of any systemic symptoms such as ptosis that manifest on the way to hospital.

Do not handle the snake with your bare hands as even a severed head can bite!

Most Traditional First Aid Methods should be Discouraged: They do more harm than good!

Release of tight bands, bandages and ligatures: Ideally, these should not be released until the patient is under medical care in hospital, resuscitation facilities are available and antivenom treatment has been started.

Tight (arterial) tourniquets are not recommended!

Patient Assessment Phase: On arrival.

Deal with any life threatening symptoms on presentation.i.e.**A**irway**, B**reathing and **C**irculation**.** Airway patency, respiratory movements, arterial pulse and level of consciousness must be checked immediately. However, the Glasgow Coma Scale cannot be used to assess the level of consciousness of patients paralyzed by neurotoxic venoms.

If there is evidence of a bite, where the skin has been broken, give **Tetanus Toxoid**

Routine use of **anti-biotic** is not necessary, although it should be considered if there is evidence of cellulitis or necrosis.

Diagnosis Phase: General Principles

- Where possible identify the snake responsible.
- **All patients will be kept under observation for a minimum of 24 hours.** Cobra produces symptoms as early as 5 minutes or as late as 10 hours after the bite. Vipers take slightly longer - the mean duration of onset being 20 minutes. However, symptoms may be delayed for several hours. Sea snake bites almost always produce myotoxic features within 2 hours so that they are reliably excluded if no symptoms are evident within this period.
- In India bite marks are of no use in identifying if a species is venomous or not.
- Determine if any traditional medicines have been used, they can sometimes cause confusing symptoms.
- Determine the exact time of the bite. This can give indications as to the progression of any symptoms.
- Ask questions as to what the victim was doing at the time of the bite.

Pain

Snakebite can often cause severe pain at the bite site. This can be treated with painkillers such as paracetamol. Pediatric dose 10mg/kg every 4-6 hourly orally.

Aspirin should not be used due to its adverse impact on coagulation. Do not use non steroidal anti-inflammatory drugs (NSAIDs) as they can cause bleeding.

Mild opiates such as Tramadol, 50 mg can be used orally or IV for relief of severe pain

Table 2: Snakes, Clinical aspects & therapeutic response

Features	cobra	Krait	Russell's viper	Sawscaled viper	Hump nose viper
Local Pain/ Tissue Damage	YES	NO	YES	YES	YES
Ptosis/ Neurological Signs	YES	YES	YES	NO	NO
Haemostatic abnormality	NO	NO	YES	YES	YES
Renal complications	NO	NO	YES	NO	YES
Response to neostigmine	YES	NO?	NO?	NO	NO

Clinical situations in which snake-bite victims might require urgent resuscitation:

Profound hypotension and shock resulting from direct

(a) cardiovascular effects of the venom or secondary effects, such as hypovolemia, release of inflammatory vasoactive mediators, hemorrhagic shock or rarely primary anaphylaxis induced by the venom itself.
(b) Terminal respiratory failure from progressive neurotoxic envenoming.
(c) Sudden deterioration or rapid development of severe systemic envenoming following the release of a tight tourniquet or compression bandage.
(d) Cardiac arrest precipitated by hyperkalemia resulting from skeletal muscle breakdown.
(e) If the patient arrives late: Late results of severe envenoming such as renal failure and septicemia complicating local necrosis.

Early clues that a patient has severe envenoming:

- Snake identified as a very dangerous one.
- Rapid early extension of local swelling from the site of the bite.
- Early tender enlargement of local lymph nodes, indicating spread of venom in the lymphatic system.
- Early systemic symptoms: collapse (hypotension, shock), nausea, vomiting, diarrhoea, severe headache, "heaviness" of the eyelids, inappropriate (pathological) drowsiness or early ptosis/ ophthalmoplegia.
- Early spontaneous systemic bleeding.
- Passage of dark brown/black urine

Investigations/laboratory tests

20-minute whole blood clotting test (20WBCT)

Place 2 mls of freshly sampled venous blood in a small, new or heat cleaned, dry, glass vessel. Leave undisturbed for 20 minutes at ambient temperature. Tip the vessel once. If the blood is still liquid (unclotted) and runs out, the patient has hypofibrinogenaemia ("incoagulable blood").

If the vessel used for the test is not made of ordinary glass, or if it has been cleaned with detergent, its wall may not stimulate clotting of the blood sample (surface activation of factor XI–Hageman factor) and test will be invalid

If there is any doubt, repeat the test in duplicate, including a "control" (blood from a healthy person such as a relative

Other tests

Hemoglobin concentration/ hematocrit: A transient increase indicates hemoconcentration

Platelet count: This may be decreased in victims of envenoming by vipers.

White blood cell count: An early neutrophil leucocytosis is evidence of systemic envenoming from any species.

Blood lm: Fragmented red cells ("helmet cell", schistocytes) are seen when there is microangiopathic haemolysis.

Plasma/serum: May be pinkish or brownish if there is gross hemoglobinaemia or myoglobinaemia.

Biochemical abnormalities: Aminotransferases and muscle enzymes (creatine kinase, aldolase etc) will be elevated if there is severe local muscle damage. Bilirubin is elevated following massive extravasation of blood. Potassium, creatinine, urea or blood urea nitrogen levels are raised in the renal failure of Russell's viper, hump-nosed viper bites and sea snakebites. Early hyperkalaemia may be seen following extensive rhabdomyolysis in sea snake-bites. Bicarbonate will be low in metabolic acidosis (e.g. renal failure).

Arterial blood gases and pH may show evidence of respiratory failure

Warning: Arterial puncture is contraindicated in patients with hemostatic abnormalities (Viperidae)
Desaturation: Arterial oxygen saturation can be assessed non-invasively.
Urine examination: The color of the urine (pink, red, brown, black) should be noted and the urine should be tested by dipsticks for blood or hemoglobin or myoglobin. Red cell casts indicate glomerular bleeding. Massive proteinuria is an early sign of the generalized increase in capillary permeability.

Antivenom treatment
Antivenom is the only specific antidote to snake venom.

What is antivenom?
the Indian antivenom manufacturers' "polyvalent anti-snake venom serum" is raised in horses using the venoms of the four most important venomous snakes in India (Indian cobra, Naja naja; Indian krait, Bungarus caeruleus; Russell's viper, Daboia russelii; saw-scaled viper, Echis carinatus),

Indications for antivenom

Systemic envenoming:
Hemostatic abnormalities: Spontaneous systemic bleeding (clinical),
Coagulopathy (20WBCT or other laboratory tests such as Prothrombin time) or thrombocytopenia (<100 x 109/litre or 100 000/cu mm) (laboratory).
Neurotoxic signs: ptosis, external ophthalmoplegia, paralysis etc (clinical).
Cardiovascular abnormalities: hypotension, shock, cardiac arrhythmia (clinical), abnormal ECG.
Acute kidney injury (renal failure): oliguria/anuria (clinical), rising blood creatinine/ urea (laboratory).
(Haemoglobin-/myoglobinuria:) dark brown urine (clinical), urine dipsticks, other evidence of intravascular haemolysis or generalised rhabdomyolysis (muscle aches and pains, hyperkalaemia) (clinical, laboratory).
Supporting laboratory evidence of systemic envenomings.

Local envenomation
Local swelling involving more than half of the bitten limb (in the absence of a tourniquet) within 48 hours of the bite. Swelling after bites on the digits (toes and especially fingers). Rapid extension of swelling (for example, beyond the wrist or ankle within a few hours of bite on the hands or feet). Development of an enlarged tender lymph node draining the bitten limb

How long after the bite can antivenom be expected to be effective?
Antivenom treatment should be given as soon as it is indicated. It may reverse systemic envenoming even when this has persisted for several days or, in the case of haemostatic abnormalities, for two or more weeks. It is, therefore, appropriate to give antivenom for as long as evidence of the coagulopathy persists.

Antivenom reactions
(1) Early anaphylactic reactions: Usually within 10-180 minutes of starting antivenom, the patient begins to itch (often over the scalp) and develops urticaria, dry cough, fever, nausea, vomiting, abdominal colic, diarrhoea and tachcardia.
(2) Pyrogenic (endotoxin) reactions: Usually these develop 1-2 hours after treatment. Symptoms include shaking chills (rigors), fever, vasodilatation and a fall in blood pressure. Febrile convulsions may be precipitated in children. These reactions are caused by pyrogen contamination during the manufacturing process,
(3) Late (serum sickness type) reactions: Develop 1-12 (mean 7) days after treatment. Clinical features include fever, nausea, vomiting, diarrhoea, itching, recurrent urticaria, arthralgia, myalgia, lymphadenopathy, periarticular swellings, mononeuritis multiplex, proteinuria with immune complex nephritis and, rarely, encephalopathy. Patients who suffer early reactions and are treated with antihistamines and corticosteroid are less likely to develop late reactions.
Prediction of antivenom reactions. Skin and conjunctival "hypersensitivity" tests will reveal IgE mediated. Type I hypersensitivity to horse or sheep proteins. However, since the majority of early (anaphylactic) or late (serum sickness type) antivenom reactions result from direct complement activation

rather than from IgE mediated hypersensitivity, these tests are not predictive. Since they may delay treatment and can in themselves be sensitising, these tests should not be used**.**

Contraindications to antivenom: Prophylaxis of high risk patients

There is no absolute contraindication to antivenom treatment, but

1. patients who have reacted to horse (equine) or sheep (ovine) serum in the past
2. those with a strong history of atopic diseases (especially severe asthma) are at high risk of severe reactions and should therefore be given antivenom only if they have signs of systemic envenoming.

Since no prophylactic drug regimen has proved effective in reducing the incidence or severity of early antivenom reactions, these drugs should not be used except in high risk patients.

Treatment of antivenom reactions. Early anaphylactic and pyrogenic antivenom reactions: Epinephrine (adrenaline) is given intramuscularly (into upper lateral thigh) in an initial dose of 0.01 mg/kg body weight for child. The dose can be repeated every 5-10 minutes if the patient's condition is deteriorating.

Additional treatment: an antihistamine anti-H1 blocker such as chlorphenamine maleate (children 0.2 mg/kg by intravenous injection over a few minutes)& intravenous hydrocortisone (children 2 mg/kg body weight), may prevent recurrent anaphylaxis.

Treatment of late (serum sickness) reactions: Late (serum sickness) reactions may respond to a 5-day course of oral antihistamine. Patients who fail to respond in 24-48 hours should be given a 5-day course of prednisolone. Doses: Chlorphenamine: children 0.25 mg/kg /day in divided doses. Prednisolone: children 0.7 mg/kg/day in divided doses for 5-7 days

Selection, storage and shelf life of antivenom

To retain their full potency within the limits of stated expiry dates, lyophilized antivenoms (shelf life about 5 years) should be stored at below 25ºC and liquid antivenoms (shelf life 2-3 years) should be stored at 2-8 ºC and not frozen. Ideally, antivenoms should be used before the stated expiry dates but, provided that they have been properly stored, they can be expected to retain useful activity for months or even years after these dates (WHO, 1981; O'Leary et al., 2009). In patients with severe envenoming, recently expired antivenoms may be used if there is no alternative.

Administration of antivenom

ASV is recommended to be administered in the following initial dose:

Neurotoxic/ Anti Haemostatic 8-10 Vials

Two methods of administration are recommended:

(1) Intravenous "push" injection: Reconstituted freeze-dried antivenom or neat liquid antivenom is given by slow intravenous injection (not more than 2 ml/minute).

(2) Intravenous infusion: Reconstituted freeze-dried or neat liquid antivenom is diluted in approximately 5-10 ml of isotonic fluid per kg body weight (i.e. 250-500 ml of isotonic saline)

and is infused at a constant rate over a period of about one hour.

Local administration of antivenom at the site of the bite is not recommended:

The only situations in which intramuscular administration might be considered:

(1) At a peripheral first aid station, before a patient with obvious envenoming is put in an ambulance for a journey to hospital that may last several hours;

(2) On an expedition exploring a remote area very far from medical care;

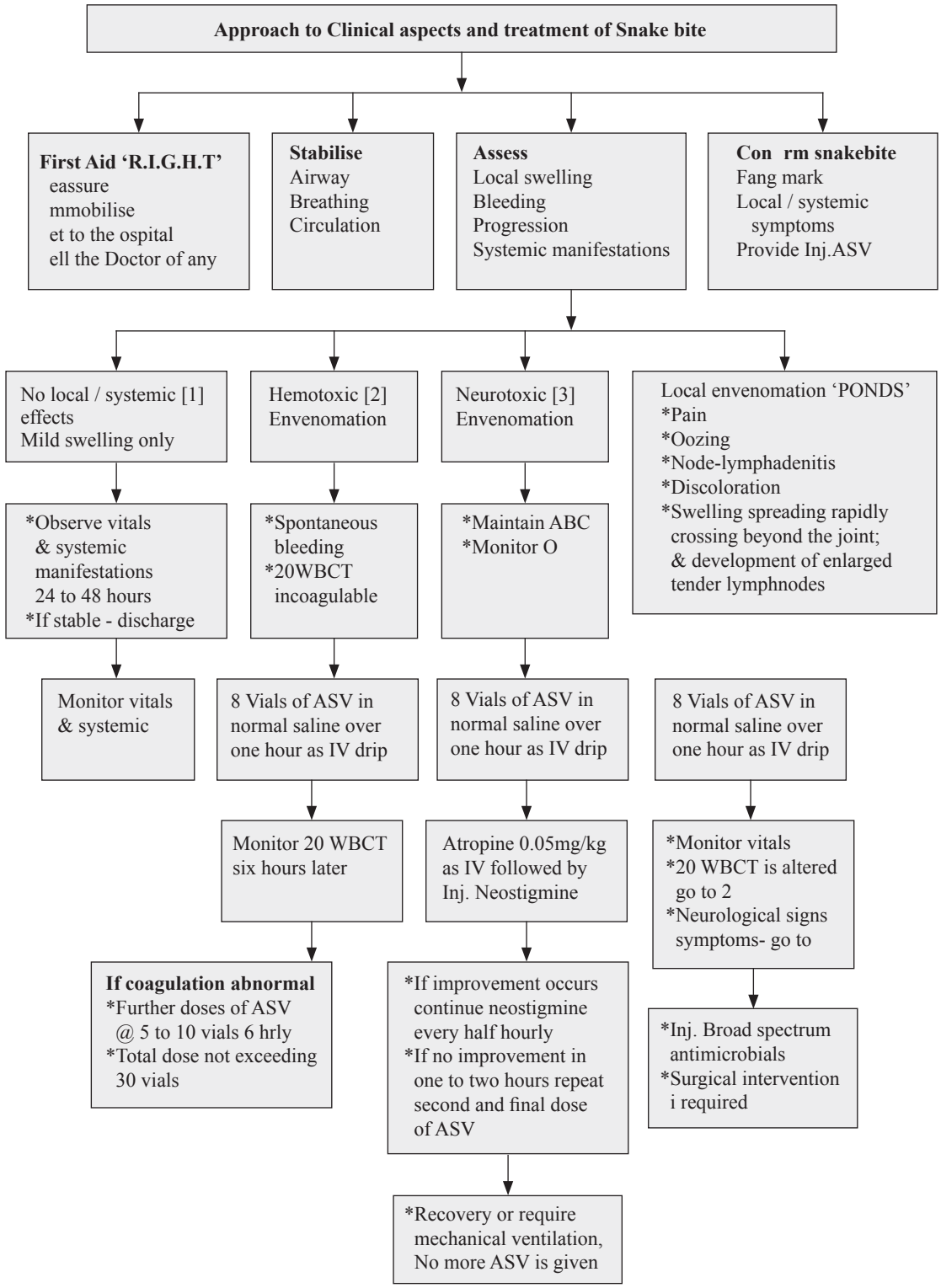
(3) When intravenous access has proved impossible.

Antivenom must never be given by the intramuscular route if it could be given intravenously.

Snakes inject the same dose of venom into children and adults. Children must therefore be given exactly the same dose of antivenom as adults.

Observation of the response to antivenom: If an adequate dose of appropriate antivenom has been administered, the following responses may be observed.

Algorithmic approach to snake bite



(a) General: The patient feels better. Nausea, headache and generalized aches and pains may disappear very quickly. This may be partly attributable to a placebo effect.
(b) Spontaneous systemic bleeding (e.g. from the gums): This usually stops within 15-30 minutes.
(c) Blood coagulability (as measured by 20WBCT): This is usually restored in 3-9 hours. Bleeding from new and partly healed wounds usually stops much sooner than this.
(d) In shocked patients: Blood pressure may increase within the first 30-60 minutes and arrhythmias such as sinus bradycardia may resolve.
(e) Neurotoxic envenoming of the post-synaptic type (cobra bites) may begin to improve as early as 30 minutes after antivenom, but usually takes several hours. Envenoming with presynaptic toxins (kraits and sea snakes) will not respond in this way.
(f) Active hemolysis and rhabdomyolysis may cease within a few hours and the urine returns to its normal color.

Recurrence of systemic envenoming
Signs of systemic envenomation may recur hours or even days after initially good response to antivenom. This has been explained by ongoing absorption of venom from the blood - which has a half life of 26-95 hours. He therefore suggests daily evaluation of patients for at least 3-4 days
The possible explanation for these manifestations is that local blebs constitute a venom depot which is suddenly released into the blood stream, especially when the wound is handled surgically

Criteria for repeating the initial dose of antivenom
Persistence or recurrence of blood incoagulability after 6 hours or of bleeding after1-2 hours. Deteriorating neurotoxic or cardiovascular signs after 1-2 hours.
If the blood remains incoagulable (as measured by 20WBCT) six hours after the initial dose of antivenom, the same dose should be repeated. This is based on the observation that, if a large dose of antivenom (more than enough to neutralize the venom procoagulant enzymes) is given initially, the time taken for the liver to restore coagulable levels of fibrinogen and other clotting factors is 3-9 hours.

In patients who continue to bleed briskly, the dose of antivenom should be repeated within 1-2 hours.

Maximum dose of ASV
Neurotoxic – 20 vials
Antihemostatic - 30 vials

Treatment of neurotoxic envenoming
Neostigmine – it is useful for neuromuscular junction blokage caused by neurotoxic snake bites. It is useful only for postsynaptic type of blokage (occuring with cobra bites) and not for presynaptic block (Krait bites cause both pre &post synaptic blokage). An Edrophonium test can predict use-fullness of neostigmine but not available in India .The response to first dose is useful. The dose is 10 to 40 microgram/kg 2-4 hrly till complete neurological recovery. It is to be given with atropine

Ventilatory support for patients with respiratory failure

Key Messages
1. History of snake bite and fang marks are not must to diagnose snake envenomation. (*e.g. Kraits & Sea snakes*).
2. Cobra, Krait, Russell's Viper & Saw scaled Viper account for majority of poisonous snake bites in India.
3. Species identification by clinical syndromic approach is quite reliable.
4. In areas known for Krait bites when a perfectly normal person, sleeping on floor reports early morning with vomiting, abdominal pain & bulbar palsy, it should be diagnosed as Krait envenomation, unless proved otherwise.
5. All snake bites do not need ASV.
6. 20 WBCT is simple, cheap, reliable & bedside tool in hands of clinician for diagnosing coagulopathy of Viper bites.
7. Don't underestimate local complications of snake bites, it may lead to gangrene & amputation.
8. Endotracheal intubation & mechanical ventilation are of utmost important in saving a child with neurotoxic respiratory paralysis in isolation & in combination with ASV.
9. Don't ignore bite by small snakes, bite after eating prey or bite after several strikes – all such bites are capable of severe envenomation.

10. Don't handle freshly killed snakes. Bite reflex is preserved in them & can lead to envenomation.

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References

1. Warrell DA. Guidelines for the management of snake bite. WHO; 2010:1-151
2. Indian national snake bite protocols.2007:4-26
3. Handbook on treatment guidelines for snake bite and Scorpion sting Tamil Nadu Health Systems Project Health and Family Welfare Department Government of Tamil Nadu, Chennai. 2008; 1-56
4. Bawaskar H S , Bawaskar P H. Management of Snake bite and Scorpion Sting. Quarterly medical review 2009 Oct-Dec; 60, No. 4
5. Punde DP.Management of snake bite in rural maharastra: A 10 yr experience. The National Medical Journal of India. 2005;18.no.2
6. Mathew JL. Snake –bite Pediatric Today. 1999;ii:36-44.
7. Warrell DA. The clinical management of snake bites in the Southeast Asian region. Southeast asian journal of tropical medicine and public health 1999;30:1-85.
8. Warrell Da. Venoms, toxins and poisonons of animals and plants. In; oxford Textbook of medicine, 3rd edn, weatherall DJ, lendingham J.G.G and warrrell Da (editors). New york : Oxford University Press 1996 Pp.1124-40.
9. Bawaskar HS, Bawaskar BH. Profile of snake bites envenoming in western Maharashtra, India.
Trans Roy Soc Trop Med Hyg 2002; 96: 79-84.
10. Bawaskar HS. Snake bite and scorpion stings.In; Khubchandani R,Gajendrsgadkar A, Bavdekar SB, Shah NK. [Eds] Frequently asked questions Ask IAP: a series. Basics and Beyond. IAP Action Plan 2006; 109-11

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