Review:
Management of Snake Bite and Scorpion Sting
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CONTENTS

1. Snake bite .................................................................................................................. 4
2. Scorpion Sting .......................................................................................................... 26
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Snake bite

Snake bite is an acute life threatening medical emergency often faced by farmers and farm labourers. Early diagnosis of envenoming by venomous snake and its rational and accurate management may save life. Rural Indian victims of snake bite are reported earlier due to easily available transport including auto and jeeps and constructed approachable roads to the majority of villages. Irrespective of early reporting, the fatality in venomous snake envenoming is due to non-availability of medical officers at primary health Center (PHC), inadequate facilities including anti-snake venom (ASV), and resuscitation trolley: working laryngoscope, endo tracheal tubes, Ambu bag, ventilator and other emergency medicine. Many times the medical officer is a freshly passed graduate and has not seen and treated the venomous snake bites before. This adds to the morbidity and mortality. Because of expensive ASV and it not being easily available to private hospitals, many doctors avoid admitting the case due to threat of anaphylaxis. Moreover the poor rural population cannot afford expensive ASV and treatment at private hospitals. Snake bite should be declared as an occupational hazard. ASV should be available free of cost to victims admitted to private hospitals. ASV is always in short supply. To avoid the crisis of ASV supply, peripheral doctors should be trained regarding management of snake bite and indications of ASV. Availability of snake venom antigen detection kit (ELISA Mono-specific) is a must. Antivenin producers in India should be encouraged to prepare antivenom from venom obtained from snakes caught from relevant areas of the country.

Introduction

Venomous snake bite is an important public health hazard in tropical and subtropical countries. In rural areas snake bite poisoning is a leading cause of premature death of young earning member of the family. 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. Moreover snake bite is not a notified disease in medical fraternity. It is surprising that, snake bite poisoning is seldom mentioned as a priority for health research in a developing country like India. The grant allocated for snake bite is many times less than the grant allocated to amoebic dysentery (with a negligible fatality as compared to snake bite). Unfortunately public health authorities, nationally and internationally, have given little attention to this grave, life threatening medical problem, relegating snake bite envenoming to the category of a major neglected disease of the 21st century. There should be more encouragement from government and other funding agencies for conducting research. Moreover there are very few medical scientists taking interest or carrying out research in this field.

Most of the venomous species of snakes are “sit and wait” predators wherein they lie camouflaged lying in wait for their potential victim and they strike when the prey comes within their striking distance. The snake usually then lets go allowing the venom to take effect after which they follow their prey by following its scent trail. So after a human strike, it is very likely that the snake will be found in the 30 foot radius and it should be remembered that they are no less dangerous after the first strike. So victim should be moved away from the area.
Etiology
There are about 216 species of snakes identifiable in India, of which 52 are known to be venomous. The major families of poisonous snakes in India are Elapidae (Cobra *Naja naja*, king cobra and kraits) viperidae (*Russell’s viper*, *Echis carinatus* or saw scaled viper or carpet viper and pit viper) and hydropidae (sea snake). 7

Snake Venom
Snakes are highly specialized animals. Their gut secretes powerful fast acting digestive enzymes. A pair of salivary glands secrete a powerful multipurpose enzyme fluid that flows at the time of envenoming through fine channeled or grooved teeth called fangs. Venom immobilizes the prey and facilitates its swallowing. It is quite clear that snake venom is not a substance evolved to attack man or any big vertebrates. A snake can bite and continue to secrete venom a number of times in succession. Dr PJ Deorus from India studied the average venom yield per bite in venomous snakes

<table>
<thead>
<tr>
<th>Venom Type</th>
<th>Venom Yield</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cobra</td>
<td>0.2 gram of the dry weight of lyophilized venom.</td>
</tr>
<tr>
<td>Krait</td>
<td>0.022 gram.</td>
</tr>
<tr>
<td><em>Russell’s viper</em></td>
<td>0.15 grams</td>
</tr>
<tr>
<td><em>Echis carinatus</em></td>
<td>0.0046 grams.</td>
</tr>
</tbody>
</table>

Lethal dose of these venoms for man has been reported

<table>
<thead>
<tr>
<th>Venom Type</th>
<th>Lethal Dose</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cobra</td>
<td>0.12 grams</td>
</tr>
<tr>
<td>Krait</td>
<td>0.06 grams</td>
</tr>
<tr>
<td><em>Russell’s viper</em></td>
<td>0.15 grams</td>
</tr>
<tr>
<td><em>Echis carinatus</em></td>
<td>0.08 grams.</td>
</tr>
</tbody>
</table>

One ml of polyvalent antivenin neutralized

<table>
<thead>
<tr>
<th>Venom Type</th>
<th>Neutralized</th>
</tr>
</thead>
<tbody>
<tr>
<td>Cobra</td>
<td>0.6 mg</td>
</tr>
<tr>
<td>Krait</td>
<td>0.45 mg</td>
</tr>
<tr>
<td><em>Russell’s viper</em></td>
<td>0.6 mg</td>
</tr>
<tr>
<td><em>Echis carinatus</em></td>
<td>0.45 mg.</td>
</tr>
</tbody>
</table>

(Anti snake venom)ASV has a half life of 26-95 hours (8,9)

Venom secretion in all venomous snakes appears to vary in seasons. In warmer months the output is more than in the cold season. Similarly darker the snake it secretes more venom as compared to a light colored snake. While the venom is viscous and comes out in small quantity in winter and in light colored snake. This explains the high fatality rate seen during summer and August, September and October months due to high environmental temperature. Most snakes inject 10% of the available venom in a single strike. Exception is the *Russell’s viper* which injects 75% of stored venom in one bite and is responsible for high morbidity and mortality India. Venom is a cocktail made of 20 or
more components. It contains proteins, in form of enzymes, non-enzymatic polypeptide toxins and non-toxic nerve growth factors. Enzymes are digestive hydrolase’s, hyaluronidase and various activators and inactivators of physiological processes. Majority of venoms consist of l-amino acid oxidase, phospho mono-and diesterases, 5’nucleotidase, DNAase, phospholipase A2, peptidase and NAD-nucleosides. Krait and cobra venom also contains acetylcholine esterase, phospholipase B and glycerophosphatase as against the vipers venoms which have endopeptidase, arginine ester hydrolase, kininogenase and thrombin like (Echis carinatus), factor X and prothrombin activating enzyme (Russell’s viper) Lecithinase. Phospholipase A2 is seen in majority of venoms extensively studied. It destroys mitochondria, red blood cells, leucocytes, platelets, peripheral nerve endings, skeletal muscle, vascular endothelium and other membranes, produces presynaptic neurotoxic activity (krait), opioid–like sedative effects and autopharmacological release of histamine (anaphylaxis). The acetylcholine esterase found in most krait and cobra venom does not contribute to their neurotoxicity. Hyaluronidase promotes the spread of venom through tissue. Proteolytic enzymes (endopeptidases or hydrolases) are responsible for local changes in permeability leading to edema, blistering and bruising and local necrosis. Severe, irreparable local tissue loss due to cobra venom is due to myocytolysis. Cobra venom is rich in postsynaptic neurotoxins called alpha-bungarotoxin and cobra toxin. The acetyl choline receptors are primary signal transducers at the neuromuscular junction. It is a multi-subunit, intrinsic membrane protein. Both short and long acting toxins from cobra venom bind specifically to acetyl choline receptors, preventing the interaction between acetyl choline and receptors on postsynaptic membrane. This action subsequently prevents the opening of the sodium channels associated with acetyl choline receptors and is responsible for neuromuscular blockade. Treatment with appropriate anti-venom can result in rapid reversal of paralysis. It is suggested that anti-venom accelerates the dissociation of the toxin-receptors complex, which leads to a reversal of paralysis. Krair venom in India contains both pre-synaptic beta bungarotoxin and pre-synaptic alfa-bungarotoxins. These toxins initially release acetylcholine at the nerve ending at neuromuscular junction and then damage the nerve ending subsequently, prevent the release of neurotransmitter acetyl choline. This explains the acute abdominal colicky pain with salivation, vomiting and “gooseflesh” in krait bite (premonitory signs and symptom of krait bite). Envenoming by kraits is associated with a syndrome of neuromuscular paralysis that falls into three distinct phases. The first phase is a rapid onset phase leading to profound paralysis within 30 to 60 minutes. The second is a stable phase of deep paralysis lasting 2 to 3 days. The third is a recovery phase 2 to 3 weeks. This explains the prolonged period of ventilator support and intensive care requirements essential for recovery. Neuromuscular blockade by the short chain neurotoxin (cobra toxin, alpha bungarotoxin) is more readily reversible than that with long chain toxins (alpha–bungarotoxin). Beta bungarotoxin in the krait venom bears similarity to botulinum toxin. The venom of cobra and krait is of smaller molecular size and is rapidly absorbed into circulation. This is the reason why a victim of cobra bite can die within 8 minutes. Absorption is further accelerated by threat of death and liberated catecholamine and running. Cobra unlike the krait deposits it venom deeply. This in combination with hyaluronidase, allows spreading of the venom to occur rapidly and symptoms to arise abruptly. Interestingly, this rapidity of onset prompts the rural victim in India to seek care quickly after the cobra bite. While more insidious onset prompts the rural victim in India to seek care quickly after the cobra bite.
(mantrik or tantrik) for natural curatives. Krait venom is ten times more lethal than cobra but the victim reports too late (due to delayed absorption of venom as it usually bites a person sleeping on the floor and it does not cause local effects, moreover reflexes in the sleep are blunted.) The smaller sized fangs usually inject the venom skin deep where there is poor circulation. Cardiotoxin contents of cobra venom are extremely lethal to myocardium. Cardio-toxin has direct action on skeletal, cardiac and smooth muscles, nerves and neuromuscular junction and is responsible for paralysis, circulatory, respiratory failure, bradycardia, heart block and systolic cardiac arrest by releasing calcium ions from the surface membrane to the myocardium.

Arginine –ester-hydrolase content of viper venom causes coagulation and release of bradykinin responsible for sudden hypotension and anaphylaxis. It is similar to thrombin in its action. Viper venoms interfere with blood clotting. This has been extensively studied. The prothrombinase causes calcium dependent conversion of prothrombin to thrombin. Venoms exhibit both anti-coagulant and coagulant effects on blood clotting mechanism resulting in defibrination syndrome or disseminated intravascular fibrino-coagulopathy. The acute bleeding is due to hypocoagulopathy or incoagulability due hypofibrinogaenaemia as a result of massive consumption of fibrinogen and fibrinolysis of blood clots. Microangiopathic haemolysis associated with disseminated intravascular coagulation, acute renal failure and hypotension (due to acute tubular necrosis) occur with Russells viper venom. Russell’s 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 Russell’s venom contains several different ‘pro-coagulants’ which activate different steps in the clotting cascade. The fibrinolytic activity of the viper venom is so fast that sometimes within 30 minutes of the bite, the coagulation factors are so depleted that blood does not clot. Russell’s viper venom activates the clotting system of the snake’s natural prey with such speed that Macfarlane was “left feeling it is almost too clever to be true”.

A protein in Echis Carinatus (saw scaled viper), found all over India except in Bengal and Kashmir has the unique effect of enhancing fibrinolysis by plasminogen activation by urokinase. Haemorrhagins -1and 2 and metaloendopeptidase cause acute rapid bleeding in brain, lungs, kidney, heart and gastrointestinal tract. It causes severe vasoconstriction followed by vasodilatation of the micro-vessels. It cause endothelial gaps due to disintegration of the endothelial cells with intracellular edema, swollen mitochondria, and dilated endoplasmic reticulum and separation of intercellular junction of the endothelial cells and local loss of basement membrane of the vessels leading to capillary and venous hemorrhage.

Cobra

All Asiatic cobras can be considered as part of a single species, *Naja naja*. Four species are found in India. *Naja naja* is seen throughout the country, *naja kauthia* in east and north east, *Naja axiana* flourishes in the extreme north west and *naja sagittifera* often seen in the Andaman
islands. Most adult cobra measure 100-150 cm, occasionally species of 210-220 cm are seen (but it is very rare). The Indian spectacled cobra is very variable in color. Often the color of cobra matches with the soil of the regional areas as a natural gift for protection from human enemies. It can be of grey, yellowish, tan, brown, reddish or black. It is easily recognized by its hood and can raise the hood more than 50% of its length. According to hood marks it is named as ‘spectacled’ with two marks or ‘monocled’ with one mark.

Cobra can be found in a variety of habitats including agricultural areas in sugarcane, paddy, soybean or Jawar growing crops. Many times in Maharashtra many cobra bite cases are reported in residence of old mud houses, huts, recently built houses, blindly handling the rubble in the attic, fire wood, dry cow dung. It is diurnal in habit. This species is shy and always attempts to escape, if it feels threatened. It produces a loud, hollow sounding, explosive hiss. It generally bites only as a last resort or it may just strike with mouth closed “head butting” its opponent. Many bites result in only little or no venom injected - “dry bite”18. It feeds on small snakes, frogs, rats and lizards. Many times it enters in the cages of hens often kept near the corner of the hut. Rats flourish in and near the grain bags in farms and grain shops. Cobras follow the rats to hunt them and accidentally bite humans handling the bags. Jawar (Sorghum) breads or chapattis are kept in the small round baskets in the small open window in the mud walled villages, where rats can enter in the basket and the cobra may follow them and bite the house wife accidentally when she blindly puts in her hand to lift up the bread. 

Snake bite cases have increased recently in Mahrashtra due to long periods of electric load shedding in villages18,19. Fangs of cobra are fixed and immobile1. Bite without envenoming is dry (defence), bite with envenoming is called Professional bite.

**A Cobra found in school bag has been reported and child died at Cobra bite.

Clinical manifestations

**Instantaneous death** is due to thought or fear of threat of death. There is sudden pouring of endogenous catecholamines resulting in cardiac (Ventricular) arrhythmias. Acute myocardial infarction and cardiac arrest in such situation may occur just on sighting the hooded cobra and can cause such an effect even without a bite11. Anxiety, tachycardia accelerated rapid absorption of venom into circulation precipitates myocardial depression, heart block, bradycardia and cardiac and respiratory arrest12,20,21. This phenomenon is not seen in children as they are unaware of death11.
**Local effects.** Cobra bite usually occurs during the day and early darkness. Common site of the bite is the extremities. Soon after bite the victims experience severe local pain, sudden development of swelling, bleeding from fang marks and subsequently clotted blood is seen over fangs abrasions. Local ecchymoses develop. Venom is a rich source of cytotoxins resulting in severe extensive edema followed by necrosis and wound takes a long time to heal, at times leaving big scars with contraction. Early skin grafting prevents the contraction and subsequent consequences. The victim may develop severe local necrosis without systemic involvement\textsuperscript{11,22}.

**Systemic involvement**

Foremost neurological manifestations include blurring of vision due to paralysis of ciliary muscles of eyes and loss of accommodation. Signs of gradual development of bulbar palsy include difficulty in deglutition, nasal twang in voice due to palatal palsy, broken neck sign i.e. unable to lift the neck from pillow. Suffocation, partial or total ophthalmoplegia and ptosis also may occur. Pupils are at times dilated and not reacting to light. The victim may suddenly lapse into an acute respiratory paralysis and shock.(11,14,19, 23,24,25).

**Locked in syndrome** - Few cases develop quadriplegia with total ophthalmoplegia 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.(26).

**Management** - The victim should not be allowed to walk or run. The bitten part should be kept below the heart level; no time should be wasted in search of the snake or application of tourniquet. No local incision must be made, sucking, application of ice or any chemical must be avoided. Only wound surface venom can be removed by clean cloth or tissue paper. The victim should be given assurance that the good treatment of snake and antidote to the bite is available at hospital. And the victim should be informed that all snakes are not poisonous and even poisonous snakes many a times do not inject venom. Time should not be wasted by taking the victim to mantrik, tantrik or village healer or temple or herbal remedies. The Victim should be removed at the earliest to the nearest primary health center by any available vehicle or even over the back of a healthy person. If the victim is transported on a motor bike (as may happen in villages) there must be another person behind him to support him, as sudden paralyses may result in fall from a speedy motor bike. At the hospital, the victim must be examined rapidly for any development of paralysis, vital functions and local site. If the snake has been killed the specimen brought should be identified or if required, the services of a snake catcher may be used.

**Anti-snake venom** (ASV) – To prepare ASV injection, dry powder in the ampoule of ASV is dissolved by adding 10 ml of distilled water as diluent. Each vial should be examined after dilution.
If it is turbid or some precipitate remains at bottom it should be discarded, (as allergic proteins may cause severe anaphylaxis).\textsuperscript{1,22}

**Test dose** – A skin test dose need not be given as there is no guarantee that non-sensitive victim will not develop reaction or otherwise. Unnecessary time is wasted in testing the ASV.\textsuperscript{1,8} If not contraindicated 0.3 ml of adrenaline can be given subcutaneously as prophylaxis against anaphylaxis before injecting the ASV.\textsuperscript{27} 100 (10 ampoules) ml of ASV to be added to 200 ml of normal saline and given by intravenous route over 30-50 minutes. The physician should sit by the side of victim to detect early signs and symptoms of anaphylaxis. 50 ml of ASV is repeated if there is no improvement in neuropahtysis - mixed 50 ml in 500 ml of normal saline and infused over 24 hours by micro drip. Administration of ASV by bolus should be avoided, as it may rapidly activate the complement system and can cause severe anaphylaxis. The ASV will neutralize the venom that is slowly absorbed from the bite site. Hypotension and bradycardia are treated with atropine or isoprenaline drip. In case of shock, without cardiac block dopamine drip may be needed.\textsuperscript{4}

Decrease in oxygen saturation or if the victim complaints of suffocation with loss of chest expansion, decreased or poor expiratory nasal blow easily felt by the dorsum of hand, reduction in one breath counting, reduction of muscle power grade 3/5, broken neck sign and pulling of saliva are indications for endotracheal intubations and ventilator support by the ambu bag or ventilator if available. At the site of bite, if the victim develops respiratory paralysis mouth to mouth respiration can be given.
Neostigmine – 25 micrograms/kg neostigmine is given by intravenous route over the first hour and then 50 micrograms/kg over next four hours can be repeated as per recovery. It can be administered as 0.5 ml neostigmine every 30 minutes till recovery occurs. It usually requires 5-8 doses. Atropine must be given just to counteract the muscarinic action of neostigmine (salivation and secretion). Neostigmine can be given by subcutaneous route.

Reaction to antivenin- Anti-snake venom is a foreign protein and can cause mild to severe reaction. It can occur between 10 to 180 minutes of administration of ASV. Mild reaction is characterized by vomiting, flushing of face and skin, hot flushes from ear and forehead, itching over scalp, and groins, urticaria, bronchospasm, cough, tachycardia, fever and palpitations. Severe reaction – ASV can cause sudden collapse with hypotension, shock low volume thready pulse, disorientation, severe bronchospasm, cyanosis, cold extremities, swelling of tongue & lips and difficulty in breathing and tracheal tug sound with angio oedema. Blood pressure may be 70 to non recordable. The incidence and severity of these reactions “are directly proportional to the speed and quantity with which the healing antiserum reaches target” (Lancet 1980).

Early reaction or immediate hypersensitivity reaction represents type IgE mediated hypersensitivity to horse serum.

Treatment
Immediately the reaction is treated with adrenaline 0.3 to 0.5 ml (300 to 500 microgram) given by intramuscular route. It can be repeated within 3-5 minutes if necessary. Intravenous fluid, aminophylline, antihistamine, and H2 blocker and hydrocortisone 100 mg are also given. If methyl prednisolone is available, it can be given to tide over the emergency.

In case of severe anaphylaxis with poor circulation intravenous adrenaline may be given in a dose 100 microgram (1000 microgram i.e. one ml of adrenaline added to 9 ml of saline so one ml contain 100 microgram adrenaline), can be repeated every 5 minutes till there is improvement in circulation and blood pressure.

Delayed reaction – may occur in the form serum sickness. It is seen after 5 to 24 days characterized by fever, arthralgia, polyarthritis, radiculopathy (mono neuritis multiplex) or Guillain-Barre syndrome, lymphadenopathy, amyotrophy. It responds to oral steroids. Serum sickness depends upon the dose of ASV administered.

Local wound- Mouth of the snake is contaminated with pathogens such as Staphylococcus and Clostridium tetani. Hence local wound must be treated with antibiotics. The victim should be immunized for tetanus. Local necrosis with gangrene needs debridement. All victims should be investigated for diabetes mellitus.
Showing cobra bite to snake catcher.

<table>
<thead>
<tr>
<th>Age/sex</th>
<th>Snake</th>
<th>Bite to Hospital in minutes</th>
<th>Total ASV in ML</th>
<th>Clinical</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>35F</td>
<td>Cobra</td>
<td>30</td>
<td>---</td>
<td>Fangs marks only</td>
<td>Dry bite</td>
</tr>
<tr>
<td>35M</td>
<td>Cobra</td>
<td>30</td>
<td>100</td>
<td>Blurred vision. Fangs marks with blood clot Local swelling+ Pain +</td>
<td>No progression to neuroparalysis</td>
</tr>
<tr>
<td>53M</td>
<td>Cobra</td>
<td>45</td>
<td>50</td>
<td>Fangs++ Swelling++ Ptoisis++</td>
<td>Ventilator -1 day</td>
</tr>
<tr>
<td>28/M</td>
<td>Cobra</td>
<td>30</td>
<td>-</td>
<td>Fang marks only</td>
<td>Dry bite</td>
</tr>
<tr>
<td>25/M</td>
<td>Cobra</td>
<td>10</td>
<td>100</td>
<td>Fangs ++ blood clot Swelling, pain+</td>
<td>No progression</td>
</tr>
<tr>
<td>30/M</td>
<td>Cobra</td>
<td>30</td>
<td>100</td>
<td>Fang++ Blood clot, Swelling+ Pain+</td>
<td>No progression</td>
</tr>
</tbody>
</table>

**Krait**

Local names- Urdu: kala gandait, gugrathi : kala taro, Marathi:kandar or manyar or kaner Tamil: kattu viriasn; Malayalam: valla pamboo.

Krait is the most poisonous among all species of snakes seen in India. Its venom is ten times more poisonous than cobra venom. The head is slightly broader than the neck with a black eye with round pupil. Its color is glossy black, bluish gray or dark brownish black with narrow (at times paired) white bands that continue towards the pointed tip of the short tail. These bands are absent on fore body and they are replaced by white vertebral spots. Whereas in the non venomous wolf snake there are complete bands from beginning of the head and absent in the narrow long tail. Kraits are active during night hours. During day time they take shelter in termite mounds, rodent burrows, piles of brick, dry coconut, cow dung, rubble and at times in a corner underneath beddings or under pillow covers. Thus the common krait is found in the vicinity of human habitation, near wattle and daub houses, mud and small huts. It is a terrestrial snake that enters in human dwelling in search of prey. The Krait eats small kraits (cannibalism), rodents, lizards and frogs. It is oviparous. It is 1-4 feet long; it is not an active or aggressive snake. Even in pucca concrete house kraits enter through the outlet pipe of bathroom if its inlet is not packed with tight iron mesh. It is interesting
to note in our study majority of victims are Hindus and few Muslims because muslims, irrespective of place and poverty, always sleep on a cot while Hindus prefer floor bed.

**Mechanism of envenoming** - High incidence of krait bite occurs during monsoon months. Because of heavy rain the holes of rats and other rodents are filled with water. There is no grain for the rats to eat in the surrounding farm hence rats enter the house. The krait may follow to hunt for the rat. Fang of the krait is sharp and short and fixed to the upper jaw. The Krait may strike a person sleeping on the ground. Also, the snake could mistakenly identify an exposed body part as prey. Most bites occur during months of June to December when snakes may, during the course of their hunting activity, linger in a person’s bedding to take advantage of warmth therein. Majority of the cases are bitten between 11 PM to 5 AM. During sleep the reflexes are blunted and with small sharp fangs the krait injects maximum venom in a person who is in sound sleep.

**Local manifestations.** The Venom is injected in skin deep or in subcutaneous tissue. It causes little local tissue damage, little or no pain and is absorbed slowly due to poor and sluggish circulation to the skin and tissue in sleep. At times the victim may forget the local pain or may give history of ant bite, rat bite or no bite. Local mild urtcaria, swelling and pin head skin bleed may be noted. No subsequent local tissue damage is noted. (Except delayed neuropathy which also rare).

**Systemic involvement** – The Victim experiences heaviness, itching at the bite site, parasthesias and weakness in the bitten part of the body. Sudden vomiting, giddiness and pain in the abdomen occur within 10-30 minutes of the bite wrongly attributed to indigestion. Usually these symptoms are neglected and the victim goes to sleep and subsequently the venom is absorbed into the circulation. Krait venom is rich in beta bungarotoxin and irreversibly blocks the presynaptic acetyl choline receptors. In Indian krait, the venom has properties to block both pre and post-synaptic acetyl
choline receptors. Initially there is release of acetyl choline resulting in autonomic stimulation characterized by vomiting, mild sweating, gooseflesh, hypertension and staring look. The mechanism for autonomic dysfunction due to snake bite is not fully understood. It is plausibly due to the blockade of the receptor site such as presynaptic alpha-2 adrenergic receptor by krait neurotoxin thereby inhibiting the inhibition of neural mediated release of nor epinephrine with resultant sympathetic over activity. It could be also due to reduction in parasympathetic activity. After 30 minutes to 8 hours and at times even after 14 hours subsequently there is development of neuroparalysis characterized by ptosis which occurs first as levator papillae muscle is rich in acetyl choline receptor. Extra ocular muscles are quite sensitive to neuromuscular blockade by elapid venom because each motor neuron innervates only 6 to 12 muscle fibers in eye muscles, compared with large proximal limb muscles where the ratio may reach 1:2000. Blurring of vision, heaviness in eyelids followed by ptosis, dilated non reacting pupils, paralysis of facial muscles resulting in myasthenia look and loss of naso-labial fold are the other symptoms Paralysis of Neck muscles, dysphagia, difficulty in deglutition, paralysis of palatal muscles, pooling of saliva, ophthalmoplegia, suffocation, quadriplegia with presence of tendon reflexes are the other signs. The diaphragm is the last to undergo paralysis. Death is due to respiratory paralysis and asphyxic cardiac arrest or iatrogenic or hospital respiratory infection and acute respiratory distress syndrome due to artificial ventilation.

Management – The Victim usually reports late due to absence of local signs and poor transport during night. The Bite may occur in any part of the body, Usually the neck, scalp, ear lobules, chest wall, popliteal fossae, axilla etc. Application of crepe bandage if bite occurs to limbs is helpful in delaying the venom absorption and respiratory paralysis. At the hospital, initial clinical signs, muscle power, respiration, oxygen saturation and intensity of expiratory nasal blow should be noted. The ASV and respiratory care and other management is same as described in cobra bite except, in krait bite the victim requires prolonged ventilator support for 3-10 days. If there is hypertension, intravenous nitroglycerine drip is given. At times the victim may land in pulmonary edema.

Many times the victims are admitted to surgical ward with pain in the abdomen for appendicitis or sent home as labelled functional pain. Even the admitted victims may suddenly relapse and manifest acute respiratory failure and doctor may face an acute emergency for which neither he himself or the ward staff are prepared.

"Thus a person reported
in mid night or early in the morning with history that he woke from floor bed due pain in abdomen, with or without history of bite coming from village or farm and bulbar palsy should be diagnosed as krait bite and closely observed unless until proved otherwise. During transport of victim to the big hospital the doctor should accompany the patient and follow the progress. This may also help the doctor to learn and gain experience to manage the next case at his center or hospital”.

Russell’s viper

Russell’s viper (daboia, *viper russelli*) snake inhabits ten South Asian countries including Pakistan, Sri Lanka, Bangladesh, Burma and Thailand. It ranks amongst the most important causes of morbidity and mortality due to snake bite. While protecting the paddy and wheat by controlling the rodent (rats) population, it kills many farmers unlucky enough to tread on it during harvest and watering the crops especially sugarcane and horticulture plants in night, due to electricity load shedding during daytime in Maharashtra.

It is 3-5 feet long snake, body is stout and has a rough appearance. The head is Triangular and broader than the neck. Nostrils are very large. The eye has a vertical pupil. It has a short and thin tail. Brown - yellow brown, with three longitudinal series prominent, large brown, black oval round spots. The spots may have pointed ends, to form a chain like pattern or may have narrow white or cream margins. The Top of head usually has narrow inverted v shaped mark. It is nocturnal. It is often found in grassy areas, scrub jungle, forest edges, rocky hillocks, dense throne hedgerows and in and around mangroves. It preys on rodents, frogs, lizard, snakes and birds. The female produces 20-60 young (viviparous) usually around June or July. The Fangs are big and semicircular mobile and attached to upper jaw. The length of the fangs in adult snake is 16 mm and they are curved. The amount of venom injected at the time of bite is $63\pm 7$ mg. When disturbed, hisses loudly “like a pressure cooker’’ and only bites as a last resort. It is the most aggressive snake 18.

Local manifestations – soon after the bite, the victim experiences severe local pain, giddiness and may often collapse. Sudden development of rapid progressive swelling may occur within 5-15 minutes of bite. The swelling may attain a length equal to the length of culprit snake. Active persistent bleeding of cuts from the fang marks seen Ecchymoses and big blebs appear in next few days on the bitten part. Subsequent ulceration or gangrene may develop. Lymph nodes proximal to the bite become enlarged and tender. There is bruising of overlying tissues and lymphangitis. Tenderness along the Hunter’s canal is often noted, over the bitten lower limb. Because of edema of muscles and bleeding there is development of compartment syndrome characterized by swelling, pain on full passive movement, tenderness over affected muscles, hypoesthesia over the areas of the nerve passing through the compartment. Ischemic damage is common if the snake injects venom at tight compartments such as pulp space of digits or the anterior tibial compartments. Absence of swelling 2 hours after the bite exclude, the envenoming or it may be a dry bite.

Systemic involvement

Haemorrhagic manifestations - Acute bleeding is due to rapid development of DIC (Disseminated Intravascular coagulation) due to consumption coagulaopathy due to conversion of procoagulant...
to coagulant and fibrinolysis, damage of vascular endothelium and platelet abnormalities\textsuperscript{10,19}. Active bleeding is seen within 30 minutes to few hours from gums. Epistaxis occurs, skin is ecchymosed, subconjunctival hemorrhages can occur. These may be haematuria, hematemeses, bleeding in peritoneal cavity, intracranial bleed and active bleeding from postpartum uterus, active uncontrolled bleed from wound, abrasion or punctured site. Hence no intramuscular injection should be given to a viper bite patient as it may result in a big haematoma. Epigastric pain often precedes acute bleeding.

**Shock**- Immediate shock is due to sudden liberation of bradykinin in the content of viper venom with angioedema and anaphylaxis due to the venom. Subsequent shock is due to massive bleeding from gums, bleeding in peritoneum and from the uterus. Intracranial bleeding may occur.
Acute bleeding in the adrenal and pituitary gland may result in hypotension and hypoglycemia. Haemorrhagic pituitary infarction results in hypogonadism, amenorrhea, Sheehan syndrome, of loss of libido\textsuperscript{38}.

**Neuroparalysis** - Ptosis gradually occurs within 6-8 hours and persists for one week. Paralysis progresses to broken neck sign. Respiratory paralysis due to Russell’s viper bite in Maharashtra is not often reported from Vidharbha region. Respiratory paralysis due to Russell’s viper is common in southern part of India and Sri Lanka. Russell’s venom causes presynaptic neuromuscular block like krait venom and resistance to anticholinesterase. Artificial ventilatory support is required for respiratory failure\textsuperscript{19,39}.

**Table- 5-Neuroparalysis due to Russell’s viper envenoming**

<table>
<thead>
<tr>
<th>Age/sex</th>
<th>month</th>
<th>Bite To Hospital hours</th>
<th>Snake</th>
<th>20WBCT</th>
<th>Bite to Neuroparalysis in hours</th>
<th>ASV ML</th>
<th>Recovery In days</th>
</tr>
</thead>
<tbody>
<tr>
<td>17 F</td>
<td>September</td>
<td>2</td>
<td>RV</td>
<td>+</td>
<td>3</td>
<td>100</td>
<td>3</td>
</tr>
<tr>
<td>27 F</td>
<td>September</td>
<td>2</td>
<td>RV</td>
<td>+</td>
<td>4</td>
<td>100</td>
<td>3</td>
</tr>
<tr>
<td>30 F</td>
<td>September</td>
<td>4</td>
<td>RV</td>
<td>+</td>
<td>4</td>
<td>100</td>
<td>3</td>
</tr>
<tr>
<td>40 F</td>
<td>September</td>
<td>2</td>
<td>RV</td>
<td>+</td>
<td>2.5</td>
<td>100</td>
<td>Died of renal failure</td>
</tr>
<tr>
<td>12 M</td>
<td>October</td>
<td>2.5</td>
<td>RV</td>
<td>+</td>
<td>2</td>
<td>100</td>
<td>4</td>
</tr>
<tr>
<td>32 F</td>
<td>September</td>
<td>2.5</td>
<td>RV</td>
<td>+</td>
<td>2</td>
<td>100</td>
<td>7</td>
</tr>
</tbody>
</table>

**Renal failure** – The common cause of morbidity and mortality due to Russell’s viper bite is acute renal failure. 20 - 40% hospitalized cases of Russell’s viper bite subsequently developed anuria, oliguria and acute renal shut down within few hours to as late as 96 hours. Direct nephrotoxic action of venom, heavy proteinuria with red cell casts suggestive of glomerular damage and glomerular capillary fibrin deposition has been reported following Russell’s viper bite. In all snake bite cases close monitoring of urine output should be strictly observed so as to diagnose earlier or impending renal failure. The foremost sign of renal failure is bilateral tenderness over renal angles. Many times vomiting, anorexia is attributed to drug induced gastritis by the newly posted medical
officer and the victim is discharged if there is no active bleeding. Many such victims are readmitted to other hospitals in acute renal failure\textsuperscript{19}. Renal failure occurs due to the direct action of the venom on renal tubules resulting in acute tubular necrosis, interstitial nephritis, patchy cortical necrosis and hypovolemic pre-renal failure due to acute blood loss and hypotension. Early administration of ASV, mannitol and diuretic may help to delay or prevent the acute renal failure. In early phase of renal failure if urine output improves with 80-100 mg of frusemide, it indicates minimum renal damage. Renal tissue responding to diuretics will further improve with slow intravenous diuretic (frusemide or toresemide) drip. Early dialysis on the verge of impending renal failure may salvage the kidney. Many times victims are reported in full blown case of renal failure with generalized anasarca, raised serum potassium and blood urea and creatinine. Often there is conjunctival edema accompanied by renal failure. There may be a capillary leak syndrome\textsuperscript{10,40,41,42,43}.

Table -6 Renal failure due to Russell’ viper envenoming

<table>
<thead>
<tr>
<th>Age/sex month</th>
<th>Bite To Hospital Hours</th>
<th>BP</th>
<th>ASV</th>
<th>Bite To RF In hours</th>
<th>BUL</th>
<th>S.Creatinine</th>
<th>Hb</th>
<th>Dialysis</th>
<th>Result</th>
</tr>
</thead>
<tbody>
<tr>
<td>40/F/Nov</td>
<td>2.5</td>
<td>80/60</td>
<td>150</td>
<td>30</td>
<td>176</td>
<td>8.6</td>
<td>7</td>
<td>NA*</td>
<td>Fatal-3day</td>
</tr>
<tr>
<td>55/M/May</td>
<td>3</td>
<td>110/80</td>
<td>120</td>
<td>24</td>
<td>130</td>
<td>4.8</td>
<td>8</td>
<td>+++</td>
<td>Recovery -3rd day</td>
</tr>
<tr>
<td>32/M/Sep.</td>
<td>3.5</td>
<td>150/90</td>
<td>170</td>
<td>24</td>
<td>107</td>
<td>5.33</td>
<td>5</td>
<td>+++</td>
<td>Fatal -7th day</td>
</tr>
</tbody>
</table>

\*Not available

Middle cerebral artery thrombosis with cerebral infarction, acute myocardial infarction and ventricular tachycardia have been reported due to Russell’s viper bite \textsuperscript{44,45,46,47}.

20WBCT test – This is the most important simple gold standard bedside blood test. Before injecting the ASV from the same vein puncture 2-3 ml of blood is withdrawn and added to a dry new glass test tube (not washed or cleaned with detergent) kept undisturbed and observed after 20 minutes, at the end of 20 minutes tipped of the blood, if the blood did not clot it confirms hypofibrinogenemia. This test should not be repeated within 6 hours of the last dose of ASV administered, as the liver takes six hours for synthesis of coagulant factors to be replaced into circulation. 20WBCT test decides the further requirement of ASV. This is an important test for diagnosis and indicates improvement\textsuperscript{48}.

Management

Initially 100 ml of ASV must be administered on arrival in 5% dextrose over 30-40 minutes. If external bleeding persists after 30-40 minutes of administration of ASV it is repeated. Another 50 ml of ASV added to 50-100 ml of 5% dextrose can be given over 24 hours by slow drip to neutralize the venom absorbed from the bite site which acts as depot. However if the 20WBCT shows non clotting blood, further dose of ASV may be added. In addition to this the victim may
Management of Snake bite and Scorpion Sting

require blood and blood products transfusion, renal dialysis and ventilator support in case of neuroparalysis, which is rare. Local wound care with early skin grafting and mobility will prevent subsequent contractures and debility.

**Echis carinatus or saw scaled viper**

The saw scaled viper is 1-3 feet long. Head of *echis carinatus* is sub-ovate with short rounded snout. Body is cylindrical, short and stout. It has a large eye with vertical pupil. The tail is very short. The Body is covered with rough, serrated flank scales and the neck is distinctly constricted. Its color is pale brown, tawny with dark brown, brick red, gray or sand colored with zigzag patterns on back. A cruciform or trident or arrowhead type or bird foot like print mark is seen on the head. It is mainly nocturnal. The snake is mostly found in open dry, sandy, rocky plains and hills. It often flourishes in heavy rainfall areas. It rests under the rocks, behind bark, at the base of thorny plants during the day. It climbs well. It is often found on the warm road or path in night. Its prey is mice, lizards, frogs, scorpions and insects. High incidence of saw scaled viper is seen in Deogad and Ratanagiri district as there is a hot humid climate. It hibernates in the winter. The quickness with which it bites on smallest provocation with an extremely rapid strike makes it one of the most dangerous snakes. It forms a double coil in the form of figure 8 with its head in the center in a striking position (looks like *chumbal* a rough cloth with round folded cushion is kept below the heavy pot on the head to support the head and scalp. Hence lay persons in Marathwada call it as *chumbal* snake). The coils keep moving against each other and serrated keels on the flank scales produce a hissing noise by friction. It is viviparous and produces 3-15 young ones at a time. It injects 0.0046 grams of venom at the one strike.

**Local manifestations**

Soon after the envenoming within one hour the victims experience mild pain and swelling at the bite site. Fangs marks or abrasions with clotted blood are seen and no active oozing of blood occurs like that of Russell’s viper bite. Swelling gradually progresses to more than one segment. The *Echis Carinatus* venom is of a bigger molecular size and is circulated by lymphatics, hence within 60-120 minutes the victim experiences a painful regional lymphadenopathy. Untreated swelling progresses to the whole limb or even to the chest wall. Ecchymoses are seen over the bitten part or may spread over lymphatic drainage area. Acute bleeding from the gum margins or from abrasion or old unhealed wound or from venepuncture site is seen within 90-120 minutes of the bite or may
be delayed by few hours to days. At times the patient remains untreated and bleeding persists in the form of blood stained sputum, haematuria and disappears without any ASV. Such victims report a few weeks later due to severe weakness, severe anemia or non healing cellulitis is with active blood oozing. Natural immunity against the *Echis carinatus* venom develops in a cases of repeated bite by same species in endemic areas with minimum clinical involvement in subsequent bite as reported in Jammu region. Renal failure due to *echis* bite is reported from Pondicherry and Jammu areas and rarely from Maharashtra. 

**Management**

The ASV requirement in the Maharashtra is 20-70 ml (average 50) ml. However the requirement is very high up to 420 ml in Pondicherry. Prolonged defibrinating syndrome due to *echis* bite have been reported. There is controversy regarding use of heparin or low molecular weight heparin. Other management is similar to Russell’ viper bite.

**Green pit viper (Trimeresurus).** It is usually found in hill forest like Mahabaleshwar, near sea level. It often flourishes on low bushes, near stream edges. Accidental bite occurs while plucking the flowers or berries. Green pit viper cases are reported from Kerala, characterized by local edema and rarely systemic bleeding as the venom has thrombin – like effects and may cause defibrinating syndrome. Coagulopathy and renal failure due to hump-nosed pit viper snake bite have been reported from Kerala state which was previously thought to be a non- venomous snake.

**Sea snakes**

Sea snakes are seen allover the coastal region. Sea snake is accidentally handled by fishermen during fishing. Its venom content includes neurotoxin, both myotoxic and hematotoxic. Soon after the bite the victim develops headache, heaviness in tongue, sweating and vomiting. Within 30 minutes to 3.5 hours after envenoming there is generalized muscle pain, stiffness and marked tenderness over the muscles. Trismus is common. Subsequently there is generalized and flaccid paralysis. Myoglobinuria appears within 30-38 hours of bite. Myoglobinuria and hyperkalaemia due
Management of Snake bite and Scorpion Sting

Hyperlanaemia causes tented T waves, widened QRS, prolonged QTc and cardiac arrest. Antivenin against the pit viper and sea snake is not available but routine polyvalent serum may be administered.

Treatment of hyperkalemia
1. Intravenous calcium gluconate
2. Intravenous diuretics
3. Glucose–insulin drip (50ml 50% glucose and 50 units of plain insulin)
4. Salbutamol inhalers
5. Dialysis

What are reasons of high morbidity and mortality due to snake bite in India?
Delay in reporting due to attending mantrik or trantrik is one of the main reasons. Herbal administration causes gastritis. Induction of forceful vomiting in case of neuroparalytic victims due to elapid bite results in aspiration and death. Tight tourniquet may lead to necrosis and gangrene. Absence of medical officers is another common problem. Failure to diagnose the early krait bite (pain in abdomen, floor bed, unknown bite, ants or rat bite, minimum local signs).

Delay or failure to administer the ASV in adequate dose.
Non-availability of intubation facilities, non functional batteries or laryngoscope, ambu bag.
Failure of intubation by untrained medical officer.
Failure to closely monitor neurological, hemorrhagic and renal profile.
Non-availability of ventilator at rural hospital or non-functioning ventilator due to want of trained doctors.

Prevention
Fire wood, dry cow dung, cattle shed and rubble should be kept away from residential area. Old storage rubble particularly in an old house should be handled in full sunlight. Rubble in the attic should not be handled blindly. Bare foot walking in darkness, in grown grass should be avoided or one should go out with a torch and big stick so as to vibrate the place before stepping. Proper care of rats, mice and lizards must be taken. No attempt should be made to catch the snake or to kill it. Killed snake should not be handled; even sheared head of snake may inject venom. Thick electrician rubber
gloves with rubber shoes should be worn at the time of handling the Jowar (sorghum) or paddy or sugarcane husk. Everybody should sleep on a cot with mosquito net, which will prevent snake, scorpion and mosquito bites alike.

45 year old man reported within 30 minutes of cobra bite in conscious state to primary health center. Medical officer noted bulbar palsy but did not administer the anti snake venom and victim was referred to rural hospital. Victim died on way to hospital. Thus referring victim with venomous envenoming without giving ASV to big hospital contributed to their death.

Training in appropriate use of antivenin and protocol of indications for its use should be arranged at general hospital level. Mere history of snake bite should not be the indication for administration of expensive and risky anti snake venom. ELISA test of venom detection should be prepared and then monospecific antivenom will be of much more use. Neuroparalytic victim should be given semi-prone position to avoid aspiration till intubated.

Attempts should be made to prepare venomous snake toxoid to immunize the farmers against venomous snake toxins in endemic areas. Many times ASV is in short supply, it can give rise to severe life threatening anaphylaxis reaction, is expensive and needs natural resources (an animal, laboratory) for its preparation. Toxicologists should make an attempt to synthesize the pharmacological antidote to venom actions or should prepare a chemical receptors product so that the venom might attack the external injected receptors and protect the natural receptors. Anti venom producers in India should be encouraged to prepare antivenom from venoms obtained from snakes caught from relevant areas of country, Regional snake park and snake venom banks should be encouraged.

Snake bite is a life threatening unnoticed sudden onset accident like earthquake in the family of poor farmer or farm labourers. Young and earning member of farmer family is prone to this accident. Victim should be provided all possible help (money, transfer and treatment) and facilities whatever required saving his life at private or government hospital. It is our moral duty to save the honest sons of soil.

The attending doctor gets immense satisfaction when the serious poor victim of snake bite recovers. Scorpion and Snake envenoming should be included in graduate and post graduate medical training.

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Scorpion Sting

Scorpion envenomation is a public health problem in tropical and subtropical countries, especially in Africa, Middle East, Latin America and India. At times it poses a significant life threatening acute time limiting cardiovascular emergency. Irrespective of different species of scorpion similar cardiovascular effects are reported. Scientists working on this problem have tried to understand the pathophysiology of severe scorpion sting by various investigations including, neurotransmitter study, radioisotope study, echocardiography, haemodynamic pattern and clinical manifestations. Various regimen including vasodilators, antivenin, platelet activating inhibitors, inotropic support, and metabolic rectifier such as insulin and L-carnitine have been tried. Irrespective of the understanding of detailed pathophysiology and its management, the fatality remains high in rural areas due to non-approachable medical facilities and faith in village healers which delays hospitalization. Scorpion envenoming has been underestimated as this problem is faced by the majority of underdeveloped and developing countries. Moreover the medical attendee from poor countries may not be aware of western line of treatment of scorpion sting. Since the advent of prazosin therapy the fatality is dropped to <1% from 29%. Recently addition of scorpion antivenom within 1-2 hours of envenoming has hastened the recovery as compared to prazosin alone.

Introduction

Scorpion envenomation is a public health problem, common in certain areas of the world including Middle East, Latin America, Africa and India. 1,2,3. Mesobuthus Tamulus (Indian red scorpion) scorpion venom is a potent sodium channel activator 4. The clinical manifestations of scorpion envenomation appear to be secondary to activation of both the sympathetic and parasympathetic nervous system. In 2/3rd of victims, the main clinical manifestations of scorpion sting are local severe excruciating pain only, which radiates along with corresponding dermatomes accompanied by mild edema and local sweating at the site of sting. Systemic manifestations (vomiting, sweating, salivation, cold extremities, priapism hyper or hypotension, brady or tachycardia and ventricular premature beats or at times non-sustained ventricular tachycardia) are not uncommon due to envenoming by the lethal scorpion species Mesobuthus tamulus, Leiurus quinquestriatus, Androctonus mauretanicus, Buthus occitanus, Centruroides A crassicauda, Tityus zulianus Tityus serrulatus1,3,4. Similar cardiovascular...
manifestations have been reported irrespective of different species of scorpion\(^4\). Morbidity and mortality due to scorpion sting is related to acute pulmonary edema, cardiogenic shock and multi organ failure. 434 cases were studied during ten years period at the national guard hospital in Riyadh. They showed 92% had local pain, 25.6% had systemic involvement. Hypertension was seen in 17%, Tachycardia in 4% cases\(^5\). We from Western Maharashtra reported 526 cases studied between 1984-1991, of these 236 (45%) had hypertension, 27(5%) had hypertension with pulmonary edema, 139(27%) had pulmonary edema, 96(27%) demonstrated tachycardia and 28(5%) died. Similar report has been obtained from Israel \(^6\). 13223 cases were recorded in the Ministry of Health in Colina state of Mexico in the year 2000-2001, of these 49% had a mild clinical, 33.8% had moderate and 17% had severe manifestations, children are more in severe group \(^7\). \textit{Tityus Zulianus} scorpion found in the Merida state, Venezuela. It was reported that children had high fatality \(^8\). 13 out of 78 cases died due to scorpion sting a report from Mahad region\(^9\). At rural hospitals from western Maharashtra, India, 3546 scorpion sting cases reported in one year of these 542 had systemic involvement\(^10\). Similar report has been received from Pondicherry, Andhra Pradesh and Karnataka states of India\(^10,11,12\). Opinions differ regarding correct treatment of scorpion envenomation\(^7\).

Recently WHO reported that the truth of scorpion sting envenoming is not known because many cases do not seek medical attention. Moreover scorpion envenoming accidents occur in villages of tropical and subtropical countries and in many countries including India it was not a notifiable disease hence actual statistical data is scarce, moreover majority of victims go to the village healers or tantriks or quacks and remain unregistered. It has been estimated that there are approximately 1 million stings per year. In Mexico alone, 250000 scorpion stings are reported yearly, in Tunisia 40000 stings, 1000 hospital admissions and 100 deaths are reported each year.
There is a high incidence in other parts of North Africa, the Middle East Iran, India, and Latin America. In Khuzestan, Southwest Iran, scorpion stings is the fourth leading cause of death attributed to *Hemi scorpion lepturus*. In Brazil, 37000 scorpion stings and 50 deaths were reported in 2005. This incidence indicates envenoming by scorpion sting is an important, yet neglected, health issue in affected parts of the world. Scientists are more keen on treating reporting and studying the snake bite than scorpion envenoming. However, the clinical research done in tropical countries is often neglected by health authorities and unfortunately yet there is no consensus regarding management of scorpion sting similar to snake bite (WHO personal communication).

Scorpion antivenin is widely used in many countries such as Brazil, Saudi Arabia, Mexico, India, and others. The acceptance of scorpion antivenin as an effective treatment in scorpion sting is based mainly on its efficacy in experimental studies. Scorpion antivenin is no better than placebo as reported from Tunisia, Israel. The beneficial effects of antivenin in protecting victims against severe scorpion sting are still questionable.

**Scorpion**

Scorpions have been recognized by a sting with severe excruciating pain which is long lasting but rarely a threat to life. They are one of the oldest known terrestrial arthropods. Fossils of scorpions found in Paleozoic strata 430 million years old appear very similar to the present species. They have been able to survive heat, drought, can withstand freezing conditions for weeks, desert conditions and starvation for months, total immersion in water for days, this remarkable power of adaptation, makes them independent of ecological condition and gives the species an unbroken continuity. They are strictly carnivorous, feeding for the most part on insects. Scorpions are viviparous and give birth directly to young ones and sometimes the mother tries to eat the young, but more often, the young ones nibble the mother to death (cannibalism). Scorpions belong to venomous arthropods in the class Arachnida. Scorpions take shelter under barks of trees, dry firewood or cow dung, in the piles of bricks, paddy husk, beddings, loose tiles of huts at times in the shoe left empty over night and in pockets of trousers and shirt, carvings, crevices of window and doors. In a tropical country the sparrow usually brings small scorpion along with the dried grass to build a nest over the window of a pucca concrete house. Farmers and farm labourers are often stung by the scorpion during handling of paddy husk, harvesting grass over bund in the months of September to November. Travelers while walking barefoot in the desert are more prone to these painful life threatening accidents.
There are around 1400 species of scorpions but only 46-50 of these are potentially lethal to humans\(^3\). Lethal species belongs to *Androctonus* (Morocco and Senegal eastwards to India), *Buthus* (Mediterranean, middle East and east Africa), *Hottentotta* (northern Africa and middle east), *Leiurus* (East Africa and Middle East), *parabuthus* (Sudan to south Africa), *Mesobuthus* (India, Southern and central Asia), *Tityus* (south America), *Centruroides* (USA, Mexico, and central America). *Hemiscorpius lepturus* (family scorpionidae) is a dangerous species seen in Iran. *Palmaneus garvimanus* a cactoid species scorpion is of bigger size as compared to other species black in color and it causes severe pain with mild sweating \(^3,\,33\).

**Venom**

Tail end of the scorpion contains two telson glands which actively secret the venom at the time of sting which is injected into the prey by sharp stinger. All scorpion species secret venom. Venom is a mixture of various active substances, of these, neurotoxins are the most important\(^34\). Neurotoxins consist of different small size proteins with sodium and potassium cations which interfere with the neurotransmitters in the victim. Venom action on neurotransmitter is rapid and fast. It contains a peptide neurotoxin that opens the Na⁺ channels (B–toxin). Sodium is primarily an extra cellular ion maintaining electric voltage difference across the cell membrane. The Scorpion venom depolarizes the cell membrane, in addition it also inhibits the deactivation of Na⁺ channels (alpha- toxin). There is massive release of endogenous catecholamine into the circulation due to delayed inactivation of sodium neuronal channel by the venom\(^4\). Thus venom of the Mesobuthus *Tamulus* (an Indian red scorpion), *Buthus Martensi* (Chinese scorpion) and *Leiurus Quinguestriatus* (Israel scorpion) causes autonomic storm by stimulating both sympathetic and parasympathetic nervous system. Charybdotoxine another component of the venom inhibits the calcium dependent K⁺ channels, similarly iberiotoxin isolated from *Mesobuthus Tamulus* has similar action on K⁺ channels\(^35,\,36\).

The venom of *leius* species includes chlorotoxin which acts on Chloride — channels. Scorpion venom also contains serotonin, which causes local pain at the site of sting. The venom of *Tityus* species a kallikrenin inhibitor causes raised bradykinin\(^37\). Venom of *Tityus Serrulatus* from Trinidad is pancreotoxice responsible for development of acute pancreatitis. Hemi scorpion *leptirus* is the most dangerous scorpion of Khuzestan, south west, hot and humid province of Iran \(^3\). Venom causes severe local tissue necrosis, renal failure and cardio respiratory arrest\(^3\).
Scorpion sting - Clinical Features

- Local pain without systemic involvement is benign
- Vomiting, sweating, salivation, priapism in male, cold extremities suggestive of autonomic storm. Needs close monitoring
- Hypertension, hypotension, bradycardia, tachycardia, ventricular entopic and acute myocardial infarction like pattern seen in ECG
- Pulmonary edema, hypotension and tachycardia with respiratory failure seen within 30 minute to 10 hours of sting
- Massive life threatening pulmonary edema needs rapid intervention
- Tachycardia >125 per minute with warm extremities, with or without pulmonary edema with cadaver pallor with convulsions suggestive of poor prognosis.

Clinical manifestations

Clinical effects of the envenoming depend upon the species of scorpion and dose of venom injected at the time of sting. The severity of envenoming is related to age, size of the scorpion and season. High incidence of pulmonary edema and fatality is seen in the month June, September and October 3,9,38.

Irrespective of different species with few exceptions (Iran and Trinidad) the cardiovascular manifestations due to envenoming are similar1,3,6,17,24,39. The early or premonitory clinical manifestations as result of autonomic storm are characterized by vomiting 34%, profuse sweating allover body 45%, priapism in males 28%, cold extremities 71% and mild tolerable pain which becomes severe. When extremities become warm it is a sign of recovery 40.
On the basis of clinical presentations or course the hospitalized patients may be

1. Severe local pain only or grade I
2. Systemic involvement

Local pain or grade I – severe excruciating pain is the only clinical manifestation seen in 35% of cases. In 57%, 33%, 11% cases lower, upper extremities and other parts of the body are the site of sting respectively. Severe pain radiates along the corresponding dermatomes. Due to intolerable pain, inconsolable crying in a child which is of sudden onset is a diagnostic sign (especially in darkness when one can not find the culprit). Children are confused and anxious due to pain. Local edema, urticaria, fasciculation and spasm of underlying muscles are seen at the site of sting due persistent stimulation of pain conducting receptors and liberated serotonin\textsuperscript{29,30}. Due to pain there is transient bradycardia, transient rise in blood pressure and mild sweating but extremities are warm\textsuperscript{3}. Sudden tap at the site of sting induces severe pain and sudden withdrawal of the part is diagnostic of scorpion sting called TAP sign.

**Systemic manifestations**

All cases have premonitory signs and symptoms of autonomic storm. Clinical manifestations depend upon time lapse between sting and hospitalization or treatment received at periphery\textsuperscript{32}. According to clinical manifestations they are divided into grades II, III and IV. All cases had initial sign and symptoms suggestive of autonomic storm \textsuperscript{3}.

**Grade II** – Profuse sweating, hypertension or transient hypotension, Tachycardia, bradycardia, premature ventricular ectopics, and cold extremities.

**Grade III** – Hypertension, hypotension, tachycardia and pulmonary edema or Massive pulmonary edema, respiratory failure.

**Grade IV** - Tachycardia, hypotension, pulmonary edema with warm extremities called warm shock.

**Hypertension**

45% of victims with systemic involvement have raised blood pressure soon after the sting. Blood pressure ranges between 140/90 to 180/130 mm Hg. Children look agitated confused and have propped up eyes and puffy face\textsuperscript{38}. Hypertension is noted in the victim. It has been reported 15 minutes to 11 hours after the sting. Majority of the cases have headache, chest discomfort, and perioral pareasthesia.

Transient initial hypotension is due to dehydration caused by excessive sweating, salivation and
vomiting which is further aggravated by hot climatic condition of tropical and subtropical countries, while post adrenergic hypotension (24 to 36) hours is due to depletion of catecholamine due to over stimulated alpha-1 receptors \(^{32,41,42}\).

**Pulmonary edema**

Pulmonary edema occurs in 27-30% cases, with respiratory failure. Pulmonary edema develops within 30 minutes to maximum 10 hours after the sting. 8% cases are reported with an acute life threatening massive pulmonary edema. Rapid onset of pulmonary edema within two hours of envenoming is often accompanied by severe hypertension. Parasternal sustained systolic lift due to sudden rise in pulmonary pressure with right ventricular after load occurs\(^3,32\). Sudden onset of breathlessness, intractable cough, poor peripheral oxygenation, ice cool extremities, tachycardia with low volume thready pulse are present. Central cyanosis, bilateral moist rales heard all over chest, with loud summation gallops and transient systolic murmur with mitral valve incompetence auscultation over precordium are the other signs. Intractable cough, with massive expectoration of blood mixed froth from mouth and nostril, with central cyanosis, hypo or hypertension and loud death rattle sounds heard few feet away from the patient are suggestive of massive pulmonary edema\(^{43}\).

Victims reported late i.e. after 6-10 hours had persistence pulmonary edema or if treated by peripheral doctors with excessive intravenous fluids, steroids, antihistamines, atropine diuretics such victims develop hypotension, tachycardia, air hunger. Prolonged poor tissue circulation with accumulation of anoxic metabolites in the circulation resulted in paralysis of capillary sphincter (vasodilatation) and cadaver like appearance. Patients are irritable, disoriented with or without pulmonary edema suggestive of warm shock\(^{44,45,46}\).

58% who reported within 8 hours of sting had heart rate 110-200 (mean 143) per minute with mean blood pressure 60-113 (mean 85) with cold extremities with or without pulmonary edema, 42% cases reported later with marked tachycardia 140-200 (mean 165) with hypotension systolic blood pressure 50-90 mm Hg with warm extremities with or without pulmonary edema (warm shock)\(^{46}\). Reappearance of local pain at the site of sting which is mild or absent on
arrival is suggestive of recovery\textsuperscript{40}. Hemiplegia, cerebral edema, disseminated intravascular coagulation, due to scorpion sting have been reported. Fatality is high once neurological complications such as coma, convulsions, miosis, mydriasis occur\textsuperscript{47,48,49,50,51,52}.

Abdominal pain, nausea, vomiting common signs and symptoms of scorpion envenomation in older children and adults were also attributed to acute pancreatitis with raised level of plasma immunoactive cationic trypsin seen due to envenoming by \textit{Tityus Trinitatatis} and \textit{Leiurus Quinquestritus}\textsuperscript{53} and uncommon due to \textit{Mesobuthus Tamulus} envenoming. Scorpion envenoming rarely causes acute renal failure. However ill-treated, delayed reporting of a case may result in death due to multi-organs failure\textsuperscript{54}.

**Investigations**

<table>
<thead>
<tr>
<th>Leukocytosis 11,000-26,000 per/cu.mm</th>
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<tbody>
<tr>
<td>Increased in troponin 1 and other cardiac enzymes</td>
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<tr>
<td>Raised interleukin, tumor necrosis factor, platelet activating factor. Renin, angiotensin II, and urinary and serum catecholamine levels\textsuperscript{55,56,57}</td>
</tr>
<tr>
<td>X-Ray chest - showed typical picture of pulmonary edema with batwing appearance. At times unilateral distribution of pulmonary edema with air bronchogram and cardiomegaly\textsuperscript{17,44,58}.</td>
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**Electrocardiogram** (ECG) - ECG is the most important diagnostic and easily available tool in rural setting. Not a single victim with systemic involvement had normal ECG. Sinus bradycardia was seen in early hypertensive cases with heart rate of 42-60 per minute which persisted for 3-4 hours, ventricular premature contraction, couplets, transient runs of ventricular tachycardia and rarely fatal lethal ventricular arrhythmias, sinus tachycardia, injury to conducting system in the form of left anterior hemiblock, right bundle branch block, left bundle branch block, complete heart block, marked tented T waves mimicking an acute myocardial infarction pattern, ST elevated with
non Q wave infarction pattern, PQRST alternans have been reported. Subsequent broad wide base with round top T wave suggestive of delayed depolarization with prolonged QTc 450 – 650 Ms accompanied with asymptomatic bradycardia and hypotension was observed 36 -48 hours of hospitalization and persisted for next five days. T wave inversion persists for more than four weeks. Despite good clinical condition of the victim, ECG showed marked changes 59,60,61,62,63,64.

Echocardiography changes - showed poor global contractility 12-15 hours after the sting, with low ejection fraction, decreased systolic left ventricular performance and mitral incompetence. Abnormal diastolic filling persisted for 5 days to four weeks. Diminished or hypokinetic left ventricular global movement with decreased systolic function was seen in a scintigraphic study. But echocardiographically there is good correlation between clinical improvement and the return of the left ventricular wall motion towards normal58,60,61,62.

**Haemodynamics**- It is difficult to perform haemodynamic study in severely ill scorpion sting case. Karnad DR from India studied the haemodynamic pattern in a patient with *Mesobuthus tamulus* envenoming from western Maharashtra India. He reported that mild envenomation causes severe vasoconstriction and hypertension while predominant left ventricular dysfunction with normal systemic vascular resistance with pulmonary edema is seen in severe scorpion sting, however severe hypotension depends upon the fluid balance. While hypotension and shock with warm extremities occurs terminally due to biventricular dysfunction and terminal vasodilatation (warm shock). Similar haemodynamic pattern has been reported from Tunisia, Brazil and Israel58,64,65,66,67,68.
Pathophysiology

Delayed inactivation of neuronal sodium channels results in acute autonomic storm. Sudden liberation of endogenous catecholamine resulted in initial transient rise in blood pressure, bradycardia and increased vascular resistance. Alpha-1 receptor stimulation plays an important part in the pathogenesis of acute pulmonary edema due to scorpion sting. Accumulation of Calcium in the heart is caused by the action of a liberated catecholamine resulting in increased requirement of oxygen to myocardium with systolic and diastolic dysfunction. There is also experimental evidence of impaired coronary perfusion. In addition to this coronary circulation is further compromised due to raised level of renin and angiotensin II. There is no significant evidence of direct effects of venom on the myocardium. Reversible cardiomyopathy is attributed to catecholamines. Pulmonary edema is due to myocardial dysfunction. However acute lung injury pattern or adult respiratory distress like syndrome attributed to secretory or non cardiogenic pulmonary edema has been reported from Brazil. Myocardial and lung parenchyma injury is due to raised level of interleukin 6, tumor necrosis factor and kallikrenin and platelet activating factors.

Histopathology study showed accumulation of fluid in the alveoli and contraction band necrosis in the myocardium and hyaline membrane in the lung in a fatal scorpion sting case. The pathophysiology, clinical and histological pattern is similar to that of a patient suffering from pheochromocytoma.

On the basis of pathophysiology the therapeutic effort should be directed against the clinical manifestations of the over stimulated autonomic nervous system and after effects of excessive catecholamine and correction of hypovolemia.

Scorpion sting - Management

- If victim reports within hour of sting with autonomic storm if available scorpion antivenin in dose of 30 to 100 ml to be administered by intravenous route. After one hour it has negligible action to neutralize the venom. Even after giving antivenin victim should be closely monitored for possibility of development of pulmonary edema.
- Oral prazosin 250 microgram in children below 5 year and 500 microgram above five year to be administered every three hour interval till extremities are cold.
- Single dose of 20-30 mg frusemide, aminophylline, oxygen, in addition to prazosin to be given to pulmonary edema case.
- Intravenous sodium nitroprusside 3 -10 microgram/kg / minute or nitroglycerine drip 5 microgram per minute raised to 15 microgram per minute in case of massive pulmonary edema.
- Dobutamine 5-15 microgram / kg / min in case of warm shock
- BiPAP or non-invasive ventilator is useful for refractory pulmonary edema with respiratory failure.
Scorpion Sting - Management

- Repeat xylocaine for local pain to be avoided local pain can be well managed with oral NSAID, Diazepam and local cold therapy.
- Atropine, steroids digoxin, antihistamines and excessive diuretic to be avoided

Search strategy and selection criteria

We are studying and treating scorpion sting cases since 1977 till today. We have collection of articles from request reprints obtained from authors since 1977, before electronic media. Extensive search made by scorpion sting, pulmonary edema, catecholamine on pub med and Google.

Management

Scorpion sting is an unnoticed sudden onset accident. Majority of victims are healthy before the sting. There is sudden onset myocardial injury with normal sized heart and liberated free fatty acids, increased myocardial contraction where digoxin is no more beneficial. While excessive diuretics are hazardous, reduction of preload by applying rotating tourniquet to periphery did help in three out of four victims of severe scorpion sting with pulmonary edema.

Alpha blocking properties of chlorpromazine—one of the constituents of lytic cocktail is responsible in reducing the fatality in children, however out of 100 children with severe scorpion sting treated with lytic-cocktail 22 died as stated in a report from Pondicherry, India. Pethidine and antihistamine (promethazine) enhances the venom toxicity and should be avoided in scorpion sting. Insulin therapy was advocated by Waterman from Trinidad in 1938. Inotropic support is required in admitted patients with scorpion sting in a intensive care unit irrespective of treatment with insulin glucose drip. Recently Gupta V from India reported hypoglycemia in 30%, pulmonary edema in 40% and fatality in 35% victims of scorpion sting given insulin glucose drip, while in prazosin treated group fatality was 6.2%. Negative inotropic effects of calcium channel blocker (nifedipine) and beta-blocker enhances myocardial failure. Steroids enhance the necrotizing effects of circulating catecholamine and should be avoided in scorpion sting victims. Antihistamines inhibit calcium dependent potassium channels like that of Scorpion venom action and should be avoided.
In experimental pharmacokinetic studies with radioactively labeled scorpion venom given intravenously, it was observed that the half life of venom distribution and its excretion were 5.6 minutes and 6.4 hours respectively. Other similar studies using antivenin showed that the half life of distribution was 1-9 hours with the result of these studies, it is concluded that antivenin therapy was inefficient because no interaction could occur between scorpion toxin and antitoxin, justified the use of prazosin and dobutamine. IgG distribution half life was tenfold longer than that of venom which was short (32 min). In comparison to immunoglobins, venom distributes fast and achieves greater concentration with a shorter time needed to achieve its maximum concentration.

Severe clinical manifestations due to scorpion sting are alleviated in victim if the antivenin is given within one hour of sting. However delayed administration of scorpion antivenin did not prevent pulmonary edema. All the ten cases had severe cardiovascular manifestations, irrespective of administration of scorpion antivenin. Of these 5 recovered with prazosin and four required inotropic support and one died (a report from western Maharashtra India). The persistence of signs and symptoms of envenoming after neutralization of circulating venom could be explained by the inability of antivenin to neutralize scorpion toxins bound to their receptors on the sodium channel. A number of specific scorpion antivenins are available but their efficacy is uncertain. Ancillary treatment with vasodilators is crucial in severely envenomed patients. Administration of scorpion antivenin after one hour of sting did not prevent the development of pulmonary edema and cerebral edema, cardiac arrest.

Captopril, angiotensin converting enzyme inhibitor did help to alleviate the diuretic induced pulmonary edema in Scorpion sting. Though the result of captopril therapy is similar to other vasodilators, an author reported 5 deaths out of 38 studied cases treated in intensive care unit in tertiary care hospital.

In retrospective study of scorpion sting cases, Rajasekhar D et al from cardiology department from Andhra Pradesh reported the use of L-carnitine to reverse myocardial dysfunction following scorpion envenomation especially in patients with hypotension and severe LV dysfunction. Aprotinin was advocated in the treatment of pulmonary edema to inhibit the platelet activating factor. Recent study by Mangano DT et al confirmed that aprotinin is not free from toxicity and can result in acute renal failure, strokes and myocardial infarction. Moreover it is expensive, not easily available and can cause severe anaphylaxis.

**Prazosin**, a post-synaptic...
alpha blocker, reduces preload, causes left ventricular impedance without raising heart rate. It reverses the metabolic syndrome evoked due to excessive catecholamines. Prazosin is a pharmacological and physiological antidote to venom action. Three victims developed severe pulmonary edema irrespective of 5 ampoules of scorpion antivenin and recovered with oral prazosin, according to a recent report from Saudi Arabia. Similar observations are reported from Tunisia. Morbidity and mortality depend upon the time lapsed between sting and administration of prazosin, since the advent of prazosin the fatality is reduced to 1%. Massive life threatening pulmonary edema is due to severe hypertension or delayed reporting of victim to health center or if the attending doctor fails to administer prazosin or inadequate dose of prazosin (which is advocated three hourly intervals) or giving excessive diuretics, IV fluids, atropine, steroids and antihistamines. These cases should be treated with intravenous nitroglycerine or sodium nitroprusside drip. 7-10% pediatric cases developed marked tachycardia, hypotension with warm extremities called “warm shock”. This necessitates dobutamine drip.

Many toxins from scorpion venoms activate sodium channels, thereby enhancing neurotransmitter release. On this basis, Fantail et al in experimental study showed beneficial effects of intravenous lidocaine, a sodium channel blocker.

Seven young patients admitted with history of scorpion sting presented with pulmonary edema which was successfully managed with positive pressure ventilation with PEEP, cardiac support with inotropic and fluid balance, a report from Nepal.

Thus management strategy for severe scorpion sting depends upon the understanding of pathophysiology and proper diagnosis of clinical manifestations and their rational and timely interventions with appropriate therapeutic agents.

Scorpion antivenin is available for clinical use. Scorpion venom is a potent neuronal sodium channel activator resulting in transient cholinergic (vomiting, sweating, salivation, priapism, ventricular ectopic and bradycardia) and prolonged sympathetic (hypertension, tachycardia, cold extremities, pulmonary edema, hypotension, shock or warm extremities with pulmonary edema and death) stimulation. Ongoing cholinergic phenomenon is suggestive of free circulating scorpion venom, which can be neutralized by antivenin. While sympathetic stimulation suggests after effects and fatality due to sympathetic over activity. We treated 30 cases of severe scorpion sting with scorpion anti venom 30-50 ml and oral prazosin. We found that if the victim reported earlier, within 1-2 hours of sting the recovery time in a group treated with scorpion antivenin and prazosin is shorter than the cases treated with prazosin alone. But the cost of one ampoule of scorpion antivenin is more than Rs.350 and at times 100 ml (10Ampoules) of antivenin is advocated. While one mg prazosin cost is Rs 32 for ten tablets. Prazosin is easily available while scorpion antivenin is often in short supply as its preparation involves natural resources including scorpion, horse and laboratory with scientists.
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Brief resume

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RESEARCH

Published >50 papers in national and international journals The Lancet, Heart, Tropical Doctor, Transaction of Royal Society, Tropical Medicine, JAPI, Q.J.Med. Toxicon, etc. on Scorpion sting, Snake bite, Hypothyroidism, Acute Myocardial Infarction, Chloroquine toxicity; HIV, BCG vaccine, etc.

Contributed Chapter in Medicine Update.

Contributed Complete chapter on Scorpion sting in the 7th EDITION API TEXT BOOK OF MEDICINE 2003.

Written a Book - SCORPION STING published by popular Prakashan Mumbai.

Trained >30,000 peripheral doctors on management of Scorpion sting in rural areas

Invited as Guest speaker for discussion at the meeting on Scorpion envenoming held in London in 1993. Guest speaker at various national conferences, IMA conferences and CME.

Mortality due scorpion sting is reduced to <1% which was >45% all over Maharashtra.

Dr. Bawasker’s research on treatment of severe scorpion sting with the help of Prazosin is of importance. The death rate due severe scorpion sting is reduced to <1% (which was >6% before his research). He managed the cases without use of scorpion antivenom which is expensive, needs laboratory, animals and not free from severe anaphylaxis reactions. His research is helpful to not only India but also all over tropical and subtropical countries like Israel, Trinidad, Brazil, Saudi Arabia, Turkey, etc.
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