Dissertation On

COMPREHENSIVE ANALYSIS OF SNAKE BITE IN TERTIARY CARE CENTRE

Submitted in partial fulfilment of
Requirements for

M.D. DEGREE BRANCH I GENERAL MEDICINE
Of
THE TAMILNADU DR.M.G.R. MEDICAL UNIVERSITY, CHENNAI

MADRAS MEDICAL COLLEGE

CHENNAI – 600 003.

MARCH- 2008

CERTIFICATE

This is to certify that this dissertation entitled **"COMPREHENSIVE ANALYSIS OF SNAKE BITE IN TERTIARY CARE CENTRE"** submitted by **Dr. N. BABU** appearing for Part II M.D. Branch I General Medicine Degree examination in March 2008 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai. I forward this to the Tamil Nadu Dr.M.G.R. Medical University, Chennai, Tamil Nadu, India.

Additional Professor
Institute of Internal medicine,
Madras Medical College,
Government General Hospital,
Chennai – 600 003.

Director,
Institute of Internal Medicine,
Government General Hospital,
Chennai – 600 003.

Dean,
Madras Medical College,
Government General Hospital,
Chennai – 600 003.

DECLARATION

I solemnly declare that the dissertation titled "**COMPREHENSIVE ANALYSIS OF SNAKE BITE IN TERTIARY CARE CENTRE"** is done by me at Madras Medical College & Govt. General Hospital, Chennai during 2006-2007 under the guidance and supervision of **Prof.C.Rajendiran, M.D.**

The dissertation is submitted to The Tamilnadu Dr. M.G.R. Medical University towards the partial fulfillment of requirements for the award of M.D. Degree (Branch I) in General Medicine.

Place:

Date:

Dr. N. BABU
M.D. General Medicine
Postgraduate Student,
Institute of Internal Medicine,
Madras Medical College,
Chennai.

ACKNOWLEDGEMENT

At the outset I would like to thank my beloved Dean, Madras Medical College **Prof T. P. Kalaniti, M.D.,** for his kind permission to use the hospital resources for this study.

I would like to express my sincere gratitude to my beloved Professor and Director, Institute of Internal Medicine **Prof.P.Thirumalaikolundu Subramanian, M.D.,** for his guidance and encouragement.

With extreme gratitude, I express my indebtedness to my beloved Chief **Prof. C.Rajendiran, M.D.,** for his motivation, advice and valuable criticism, which enabled me to complete this work.

I am extremely thankful to Assistant Professors of Medicine **Dr Muthuselvan, M.D.,** and **Dr Basker, M.D.,** for their co-operation and guidance.

I am immensely grateful to the generosity shown by the patients who participated in this study. If at all, this study could contribute a little to relieve them from their suffering I feel that I have repaid a part of my debt.

CONTENTS

INTRODUCTION

Snakebite is an important preventable health hazard. Patients with snake envenomation present as emergencies with significant morbidity and mortality. Snakebite is one of the occupational hazards and the majority affected belongs to the productive group of the population. The resulting high fatality rate due to snake bite remains largely unnoticed and neglected by the medical profession.

While the precise figures for global snake bite epidemiology are not available, best of the estimates suggest that there are more than 2.5 million venomous snake bites annually, with greater than 125,000 deaths. The risk is highest in the tropics and West Africa, predominantly amongst the rural population. In India a large proportion of snake bites occur when people are working barefoot in the fields, or while walking at night or early morning through fields or along road side[1].

India an agricultural country looses substantial number of lives to snake bite. More than 2000000 snake bites are reported in the country annually and it is estimated that between 35000 and 50000 people die of snake bite each year in India[2]. This does not reflect the real situation as because majority of the cases are not reported. Though underreported this mortality is one of the highest in the world. The main reasons for

the underreporting are people's attitude of seeking indigenous medications in preference to modern health care facilities. Ignorance of conventional treatment of snake bite by the doctors further delays the proper treatment of the victims.

The outcome of the snakebite treatment largely depends on time delay between snake bite and ASV administration[3]. There is higher incidence of renal failure, respiratory failure, prolonged hospital stay and blood products administration in those who reported to the hospital lately[4]. Many studies reveal that renal abnormality correlates well with late onset of treatment[5]. Irrationally used ASV increases morbidity and mortality, and also increases the burden on the health budget of the government.

Community based studies with reference to demographical, sociocultural, clinical and economical implication should be carried out. With the analysis of the studies, various policy decisions can be made for revamping and revising the health projects and schemes. Sensitization of the public regarding the approach to snake bite victim, regulation of the media in reference to the dissemination of scientific information, addressing the policy makers to earmark funds for community education regarding snake bite and multipronged approach to snake bite as a whole is need of the hour.

AIMS AND OBJECTIVES

To study the pattern of snake bite with reference to demographic profile, treatment preferences, sociocultural beliefs, customs, regional beliefs, accessibility of health care, obstacles met during seeking of health care, average time delay in seeking medical care, dose of the first instituted ASV at the various primary health centers and district hospital, course of the patient in the hospital, treatment outcome and economic implication, impact of the snake bite on individual and community.

SNAKE BITE IN INDIA - A REVIEW

Snakes fascinate us more than any other animals on earth. Because people don't know much about them, snakes are misunderstood and feared. Of the 3000 species of snakes, about 500 belong to the three families of venomous snakes,

- Atractaspididae
- Elapidae
- Viperidae

Only about 200 species have caused death or permanent disability by biting humans. Bites by more than 30 species of the large family colubridae, once considered harmless, have produced signs of envenoming in man, while five of them have caused fatal envenoming[6]. Among the nonvenomous snakes, only the giant constrictors (family Boidae) are potentially dangerous to man. There have been a number of fatal attack by these snakes reported from Africa (rock python), south East Asia (especially Indonesia) (reticulate python) and South America (anaconda). Some of the victims, even adults, were swallowed.

There are over 270 different species of snakes in India. In India most of the snakes are absolutely harmless to humans while four species (cobra, saw-scaled viper, Russell's viper, and krait) are responsible for thousands of deaths each year. Indian snakes range in size form a few centimeters to almost ten meters in length. Snakes live in scorching deserts, humid forests, and cool hill range, in lakes, streams and even in the sea. The variety of colors and patterns rivals the butterflies while their grace and fluidity are unmatched in nature. Snake behavior and adaptations are endlessly exciting but the first step is able to identify them.

Snakes are classified morphologically by the arrangement of their scales (lepidosis), dentition, osteology, mycology, sensory organs, and the form of the hemipenes and increasingly by sequence analysis of DNA encoding important mitochondrial and other enzymes[7].

Legless lizards, such as slow worms, glass lizards, worm like geckos and legless skinks, may be distinguished from snakes by their eyelids, external ears, and friable tails and by the lack of enlarged ventral scales. Eels and pipe-shaped fish are distinguished from snakes by their gills and fins.

SENSES

Snakes probably don't see colour and many are near-sighted, including the vipers and all the burrowers. Tree snakes, rat snakes and king cobra all have good vision and can see you coming from quite a far away(though how far is not yet known) (Romulus Whitaker., snakes of India)

Snakes can smell with their nostrils but they rely mainly on the combination of their sensitive tongue (which picks up the odors) and the Jacobson's organ in the roof of the mouth (where the odors are "decoded")

The heat sensitive "pits" between the nostrils and the eye in the pit vipers can detect temperature change as slight as three thousandths of a degree centigrade (0.003 C). Pits are very helpful in finding warm-blooded rodents or birds or even a slightly warm frog or toad on a cool dark night. Pythons have similar infrared receptors along their upper lips.

Snakes can actually "hear" low frequency sounds in the 200 to 500 Hz range. They have no ears or eardrums but low sounds that hit the side of the skull are transferred to the inner ear through the jaw muscle and ear bone. They cannot hear sounds like talking, music or a gunshot.

Snakes are sensitive to vibrations through the ground and in this way can feel you walking past.

FEEDING

All snakes are primarily predators. Some hunt by stealth and ambush, while others actively pursue their prey. Occasionally snakes will scavenge freshly dead animals. Their preys are rodents, lizards, birds, and frogs. Snakes also eat bats, frog eggs, snails and slugs while some are snake-eaters. Some giant pythons can kill and ea t a deer or even a wild pig.

BREEDING

The sex organs of snakes are internal. Herpetologist sex snakes by using a probe. They can be also sexed by noting the thicker tail base and longer tail of the male. Most snakes have specific mating season each year (sometimes two seasons) and in a large country with many climatic conditions like India, mating seasons may vary from region to region. Males follow females by pheromone trail. During this, the males involve in ritual fight called combat dance. Most of the time combat dance is between males, but sometimes male and female can also involve in combat dance.

Approximately 25% of the Indian snakes are ovoviviparous, that is the eggs develop internally and female bear live young. Livebearers include the vine snakes, estuarine snakes, most sea snakes, most vipers and pit vipers. All the rest of the snakes are egg layers. Gestation lasts between 30-50 days. Snake eggs hatch in 60-70 days; hatching time depends on the incubation temperature.

FANGS[8]

All medically important species of snakes have one or more pair of fangs in their upper jaw. In India the medically important species are Elapidae (cobra and krait) and Viperidae (Russell's and saw-scaled viper). The fangs are venom-delivering apparatus. The delivery system may be primitive or highly developed. In primitive system, the toxic saliva is injected into the victim with rear teeth. In highly developed system (cobra, krait and viper) venomous glands produce venoms which are injected into the victim with the help of highly developed fangs either short and fixed or long and sheathed. In Elapidae and Viperidae the venomous glands are present at back of the eyes. Elapidae (cobra and krait) have relatively short and fixed proteroglyphous front fangs. Viperidae (viper, adders, rattlesnakes, moccainsm, lanceheaded vipers and pit vipers) have highly evolved long curved, hinged, solenoglyphous

front fangs containing a closed venom channel, like hypodermic needles.

MEDICALLY IMPORTANT SNAKES OF INDIA

India has some of the world's most venomous snakes and high density of human here makes snake bite a common medical problem. Accurate statistics are hard to come by, but perhaps half a million people are bitten by snakes each year in India. All snakes are protected under wild life protection act 1972. The big four snakes of India are cobra, krait, Russell's viper and saw-scaled viper.

Spectacled cobra

The biological name is Naja naja.Cobra is derived from the word "cobra de capello" (i.e., snake with hood in Portuguese). They are egg layers. Male are larger than females. They are nocturnal and diurnal. They live in rat holes and termite mounds. They raise the fore body, one third to one half of the total body length, to form the characteristic hood. They characteristically produce the hissing sound.

Krait

The biological name is Bungarus caeruleus.They are nocturnal and egg layers. Males are larger than females. They are notorious to visit

the dwelling area in search of prey and in that process bites sleeping victims. Their bites are usually painless and patient usually presents with descending neurological paralysis.

Saw-scaled Viper

Their biological name is Echis carinatus. They are nocturnal and ovoviviparous. When they are alarmed, they rub the saw scaled edges to produce the rasping sound. Females are larger than males.

Russell's viper

The biological name is Duboia russelli. They are viviparous. They produce characteristic hissing sound by expelling air through their large nostrils. Their bites produce both neurotoxicity and haemotoxicity.

IDENTIFICATION OF SNAKES

There is no simple and reliable method to distinguish venomous from nonvenomous snakes. The snake's upper jaw can be examined for the presence of fangs but it is too small in Elapidae and is folded back in sheath in the case of Viperidae. Snakes can be identified by some characteristic features specific to that particular snakes, like the hood of cobra, the hissing sound made by the Russell's viper, the rasping sound made by the saw-scaled viper and growl of the king cobra.

VENOM COMPOSITION[9]

Snakes have more complex of all the venoms. They may contain more than 20 components. More than 90% of dry weight is protein and the remaining are non protein ingredients. The protein component is mixture of

- Enzymes,
- Non enzymatic polypeptide toxins and
- Non toxic proteins.

Enzymes like hydrolases, hyaluronidase, L-aminoacid oxidase, phosphor mono diesterase, 5 nucleotidase, DNAse, NAD nucleosidase, phospholipase A2 and peptidases form the 80%-90% of viperid venom and 27%-70% of the elapid venom. Along with these, the elapid venom also contains glycerophosphatase, phospholipase B and acetylcholinesterase. In addition the Viperid venom contains endopeptidase, arginine ester hydrolase, kyninogens, thrombin like factor X, prothrombin activating enzyme. Hyaluronidase aids in the spread of the venom through the tissue planes. Proteolytic enzymes like endopeptidase and hydrolases cause local change in vascular permeability leading to edema, blistering and bruising.

Non-enzymatic polypeptide toxins are neurotoxins found especially in elapid and hydropids, namely postsynaptic alpha bungarotoxin and cobrotoxin and presynaptic beta bungarotoxin , crotoin and taipoxin. The alpha toxins block the ach receptors and beta toxins release the acetylcholine, destroy the presynaptic neuron and prevent further release of transmitter. Histamine and serotonin in Viperidae cause local pain and permeability.

Phospholipase A2 is the most widespread and extensively studied of all venom enzymes. Under experimental conditions it damages mitochondria, red blood cells, leucocytes, platelets, peripheral nerve endings, skeletal muscle, vascular endothelium and other membranes, and produces presynaptic neurotoxicity activity, opiate like sedative effects and the auto pharmacological release of histamine. The nontoxic protein is the nerve growth factor.

Carbohydrates, metals, lipids, free amino acid, nucleosides and biogenic amines like serotonin and acetylcholine form the nonprotein ingredients of the venom.

CLINICAL REATURES OF ENVENOMATION[10]

The clinical features of envenomation can be categorized like symptoms and signs caused by fear, direct action of various venom components on tissues, indirect effect such as complement activation and auto pharmacological release of endogenous vasoactive substances, effects of treatment and complication due to secondary infections.

Local swelling

Most of the poisonous snake bites cause local swelling in the form of edema, cellulitis with or without accompanying lymphadenitis. The reason for the local swelling is increased vascular permeability caused by factors including endopeptidase, metalloproteinase, hemorrhagins, membrane damaging polypeptide toxins, Phospholipase and endogenous autocoids such as histamine and serotonin. Myotoxins and cytotoxins cause local tissue necrosis. Venoms of Viperidae can cause generalized increase in vascular permeability leading on to pulmonary edema, serous effusion, conjunctival and facial edema and haemoconcentration.

Significant local swelling is the one in which the edema crosses the proximal major joint. This is considered one of the indications for administration of ASV.

Hypotension and shock

Some poisonous snake bites presents with hypotension and shock. This is part of autopharmocological syndrome caused due to vasodilating autocoids. Oligopeptides in Viperidae inhibit bradykinin deactivation enzymes and angiotensin converting enzyme leading on to hypotension[11]. Some viper venom can cause direct depression of the medullary vasomotor centre[19]. Snake handlers are sensitized to life threatening anaphylactic reactions. Hypotension is also due to leakage of plasma into the bitten limb. Gastrointestinal bleed is also one of the reasons for the hypotension shock syndrome. Vasodilatation of splanchnic circulation and direct effect on the myocardium also contributes.

Bleeding and clotting disturbance[12]

Venom procoagulants cause consumption coagulopathy and leads on to bleeding and at extreme cases DIC[21, 22]. Echis activates the prothrombin and Duboia activates factor X and V. Phospholipase is an anticoagulant and leads on to bleeding. Thrombocytopenia caused by consumption coagulopathy and direct activation of the platelet receptors and agglutination also contributes to bleeding disturbances. Hemorrhagins directly damage the vascular endothelium and leads on to

spontaneous systemic bleeding. Complement activation by alternate pathway by elapid and classical pathway by Viperidae will lead o to coagulation disturbances.

Intravascular hemolysis

Intravascular hemolysis is caused due to two reasons. First is some components (Phospholipase A2 and direct lytic factor) of the venom directly act on the RBC membrane and cause hemolysis[20]. Secondly, venom induced disseminated intravascular hemolysis causes microangiopathic haemolysis.

Renal failure[13]

Renal failure in snake bite is due to various reasons. The morbidity and mortality accompanying snake bite is mainly due to renal failure. The injudicious use of ASV may also aggravate the renal failure. Most of the time it is acute renal failure improves with dialysis, but some time may lead on to persistent acute renal failure or chronic renal failure. The following are the reasons.

- hypotension and hypovolemia due to snake bite may lead on to prerenal failure leading on to permanent damage if not intervened at earlier instance

- DIC caused by envenomation will cause alteration in blood flow to the renal tissues and also there will be accompanying ring DIC of the renal vasculature culminating in renal failure.
- Intravascular hemolysis also has pathogenetic significance in snake bite induced ARF[28,29].
- Direct effect of the venom on the renal tubular cells will lead on to toxin induces acute tubular necrosis and renal failure[14]
- Myoglobinuria and hemoglobinuria accompanying snake envenomation will lead on to precipitation of the same in the renal tubules leading to renal failure.
- Hyperkalemia due to hemolysis and myolysis leads on to renal damage.

Renal findings in envenomation are[20]

- Proliferative glomerulonephritis[24].
- Toxic mesangiolysis with platelet agglutination, fibrin deposition and ischemic changes.
- Distal tubular damage (lower nephron nephrosis) suggesting direct venom nephrotoxicity[23] .
- Bilateral renal cortical necrosis[15.]

- Acute interstitial nephritis[25].

Neurotoxicity

Neurotoxicity is manifested by ptosis (earliest sign), dysphonia, and dysphagia, weakness of the limbs, opthalmoplegia and respiratory paralysis. Loss of sense of smell is also proposed as the earliest sign but there is no enough documentation of the same. Neurotoxicity is seen in envenomation by cobra, krait, mamba and also by Russell's viper. The most common cause of death in neurotoxicity is respiratory paralysis followed by bulbar paralysis. Neurotoxicity may be the manifestation of either presynaptic or postsynaptic blockade. The postsynaptic neurotransmitter dysfunction is managed efficiently and early by administration of neostigmine (anticholineesterase drug). Snake bites are differentiated on the basis of response to neostigmine. Improvement with anticholinesterase drugs are predominantly seen in envenomation by Asian cobra, Australasian death adder, Latin American coral snake. Ventilator is the mainstay of management for the respiratory paralysis. Neurotoxic symptoms can be delayed up to 48 hours, especially in the case of krait bite.

Rhabdomyolysis[16]

Rhabdomyolysis is feature of sea snakes. This is due to the presynaptic neurotoxins of sea snakes. Several species of Viperidae (Sri Lankan Russell's viper) also causes rhabdomyolysis. Death in this category is due to bulbar or respiratory muscle failure, hyperkalemia and renal failure.

Venom opthalmia[17]

Corneal erosions, anterior uveitis, and secondary infections are some of the common complications encountered due to direct contact of venom in the eye. There are certain varieties of spitting cobra which can spit venom up to two meters.

Elapid envenomation

Two of the most common neurotoxic snakebite in India belongs to Elapidae[26]. They are krait and cobra. Krait bite usually causes minimal local effect. Sometimes the local effects are so minimal that the snake bite is not at all suspected as the patient's presents with descending paralysis without any obvious bite marks or local edema. Asian cobra bite usually causes tender local swelling, lymphadenopathy,

blistering and bullae[18]. Earliest symptom of neurotoxicity is vomiting[27]. But it is confused with the effect of local indigenous medications.

Preparalytic symptoms

The profound paralysis of neurotoxic snake bite is preceded by preparalytic symptoms like, contraction of frontalis, blurred vision, and paraesthesia around the mouth, hyperacusis, loss of smell and taste, headache, dizziness, vertigo. Hyperautonomicity in the form of salivation, conjunctival congestion and goose flesh are also manifestations of neurotoxicity[27].

Paralysis

The paralytic symptoms are heralded by ptosis and external opthalmoplegia, as the ocular muscles are more sensitive to neuromuscular blockade. The time of onset of paralytic symptoms also varies with different snakes. Earlier occurrences are seen in cobra bite (as early as 15 min) and late manifestation are usually seen in krait bites (can be delayed by 10 hours). At later instances palate and pharynx and laryngeal paralysis is seen. Pupils are usually dilated. With the onset of respiratory paralysis and hypoxia seizures and loss of consciousness can occur. Neurotoxicity is relieved by antivenin. Some of the snake bites (post synaptic neuromuscular blockade) are neostigmine responsive. The

neurotoxic snake bites can be managed with ventilator support only in the absence of antivenin or allergy to antivenin. With ventilator support only diaphragmatic paralysis improves by 1-4 days, the ocular muscles improve in 2-4 days and full motor recovery occurs in 3-7days[27].

Viperidae bite

The two most common Viperidae which cause major casualty is Russells viper and Saw scaled vipr[26]. Viperidae bite usually causes severe local reaction. Swelling of the bitten limb usually occurs in fifteen min. persistent bleeding from the fang marks is seen in certain cases. In severe cases, extravasations of fluid into the bitten limb may occur leading to hypovolaemia and shock[27]. Necrosis and regional lymphadenopathy is seen in majority of the cases. Some cases are complicated by compartmental syndrome leading on to prolonged morbidity and mortality. Absence of detectable swelling two hours after viper bite can be reliably taken as dry bite except tropical rattle, Burmese Russell. In these varieties, systemic envenomation can occur in the absence of local envenomation.

Coagulation dysfunction is the main manifestation of the Viperidae bite. Persistent bleeding (>10 min), from the fang puncture sites, venepuncture sites; old partially healed wounds are first sing of

coagulopathy[27]. Bleeding into the pituitary can lead on to SHEEHANS SYNDROME, usually caused by Russell's in Burma and India[27]. Menorrhagia, antepartum and postpartum haemorrhage, subarachnoid haemorrhage, intra cerebral haemorrhage, retroperitoneal haemorrhages can complicate the course of the clinical manifestation. Thromboses of major arteries such as cerebral, pulmonary and coronary arteries are also noted in certain cases. In severe cases microangiopathic hemolysis and hemolytic uremic syndrome are documented[30]. Consumption coagulopathy may lead on to disseminated intravascular coagulation.

Certain cases of viper bites are complicated by hypotensive syndrome. Patients presents with hypotension, tachycardia, vasovagal syncope and circulatory shock. Anaphylaxis to the venom or the antivenom may be the cause of such manifestations. The most common cause of death in Russell's is renal failure. Some cases of Russell's viper presents with combined toxicity (neurotoxicity and haemotoxicity). Usually such neurotoxic signs are non responsive to neostigmine and take long time for recovery.

Clinical course and prognosis

Local swelling is seen in two to four hours in vipers and cytotoxic cobra. Maximum swelling is seen in two to three days. Earliest systemic

symptom is vomiting. Earliest death is seen in case of elapid envenomation. Death can occur as early as one hour due to rapid absorption of venom. Some cases of neurotoxicity may need ventilator support for as much as ten days. Viperidae deaths are usually due to renal failure and it takes many days.

LABORATORY INVESTIGATIONS

20 min whole blood clotting test

Considered the most reliable test of coagulation and can be carried out at the bedside without specialist training. It can also be carried out in the most basic settings.

A few ml of fresh venous blood is placed n a new, clean and dry glass vessel and left at ambient temperature for 20 minutes. The glass vessel should be left undisturbed for 20 minutes and then gently tilted, not shaken. If the blood is still liquid then the patient has incoagulable blood. The vessel must not have been washed with detergent, as this will inhibit the contact element of the clotting mechanism.

Other useful tests depending on availability

- Haemoglobin/PCV/platelet count/PT/APTT/FDP/D-Dimer
- Peripheral smear

- Urine tests for proteinuria/RBC/Haemoglobinuria/myoglobinuria
- Biochemistry for serum creatinine/urea/potassium
- Oxygen saturation/PR/BP/postural blood pressure
- ECG/X-Ray/CT/Ultrasound (the use of X-Ray and ultrasound are of unproven benefit, apart from identification of bleeding in viperine bites.

MANAGEMENT OF SNAKE BITE IN SOUTH EAST ASIA

First aid treatment protocol

Of primary importance is the need to recommend the most effective first aid for the victims, to enable them to reach the nearest medical facility in the best possible condition. Much of the first aid currently carried out is ineffective and dangerous[31].

Recommended method for India[32]

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

This method will get the victim to the hospital quickly, without recourse to traditional medical approaches which can dangerously delay effective treatment [33].and will supply the doctor with the best possible information on arrival.

Newer methods considered inapplicable in the Indian context.

Pressure immobilisation méthod (PIM)

Pressure immobilization has gained some supporters on television and in the herpetology literature. Some medical textbooks have referred to it. They have not however, reviewed the research, nor considered PIM's applicability in the Indian context.

PIM was developed in Australia in 1974 by Sutherlan[34].He used monkeys for the demonstration of the techniques and the results were mixed. He argued that a crepe bandage and an integral splint be applied

over the wound to a pressure of 55 mm of mercury. The version used in India of a bandage alone, Sutherland argued would be ineffective.

Howarth in 1994 demonstrated that the pressure to be effective was different in the lower and upper limbs[35]. The upper limb pressure was 40-70 mm of mercury; the lower limb was 55-70mm of mercury.

Table for comparison of all the snakes[32].

Features	Cobra	Krait	Russells Viper	Saw scaled viper
Local pain/tissue damage	Yes	No	Yes	Yes
Ptosis / neurogogical signs	Yes	Yes	Yes!	No
Haemostatic Abnormalities	No	No!	Yes	Yes
Renal complications	No	No	Yes	No
Reponses to Neostigmine	Yes	No?	No?	No
Response to ASV	Yes	Yes	Yes	Yes

Anti snake venom

Anti snake venom is the treatment for snake bite. Anti snake venom in India is polyvalent i.e. it is effective against all the four common species; Russell viper, common cobra, common krait, and saw

scaled viper. There is no currently available monovalent ASV primarily because there are no objective means of identifying the snake species, in the absence of the dead snake. It would be impossible for the physician to determine which type of monovalent ASV to employ in the treating the patient. There are known species such as the hump-nosed pit viper where polyvalent ASV is known to be ineffective. in addition, there are regionally specific species such as sochurek's saw scaled viper in Rajasthan, where the effectiveness of polyvalent ASV may be questionable. Further work is being carried out with ASV producers to address this issue.

ASV is produced in both liquid and lyophilized forms. Half life of ASV is 26-96 hours[36]. There is no evidence to suggest which form is more effective and many doctors prefer one or the other based purely on personal choice. Liquid ASV requires a reliable cold chain and refrigeration and has a 2 year shelf life. Lyophilized ASV, in powder form, requires only to be kept cool. This is a useful feature in remote areas where power supply is inconsistent.

ASV administration criteria[32]

ASV is a scarce, costly commodity and should only be administered when there are definite signs of envenomation. Unbound,

free flowing venom, can only be neutralized when it is in the bloodstream or tissue fluid. In addition, anti-snake venom carries risks of anaphylactic reactions and should not therefore be used unnecessarily. The doctor should be prepared for such reactions.

In line with the W.H.O.SEARO, guidelines only if a patient develops one or more of the following signs/symptoms will ASV be administered[32]:

Systemic envenomation

- Evidence of coagulopathy: Primarily detected by 20WBCT or visible spontaneous systemic bleeding, gums etc. further laboratory tests for thrombocytopenia, Hb abnormalities, PCV, peripheral smear etc provide confirmation, but 20WBCT is paramount.
- Evidence of neurotoxicity: Ptosis, external opthalmoplegia, paralysis etc.
- Cardiovascular abnormalities: hypotension, shock, cardiac arrhythmia, abnormal ECG
- Acute renal failure: oliguria/anuria, rising blood creatinine/urea

- Hemoglobinuria/Myoglobinuria: dark brown urine, urine dipsticks, other evidence of intravascular haemolysis or generalized rhabdomyolysis (muscle aches and pains, hyperkalemia)
- Persistent and severe vomiting or abdominal pain

Local envenoming

- Local swelling involving more than half of the bitten limb (in the absence of a tourniquet) In the case of severe swelling after bites on the digits (toes and especially fingers) after a bite from a known necrotic species. If the tourniquet have been applied these themselves can cause swelling, once they have been removed for 1 hour and the swelling continues, then it is unlikely o be as a result of the tourniquet and ASV may be applicable.
- Rapid extension of swelling (for example beyond the wrist or ankle within a few hours of bites on the hands or feet)
- Development of an enlarged tender lymph node draining the bitten limb.

ASV administration[32]

In the absence of definitive data on the level of en venomation, such as provided by ELISA testing, symptomology is not a useful guide to the level of envenomation[37, 38, 39]. Any ASV regimen adopted is only a best estimate. What is important is that a single protocol is established and adhered to, in order to enable results to be reliably reviewed.

The recommended dosage level has been based on published research that Russell's viper injects on average 63 mg SD 7 mg of venom[40]. Logic suggests that our initial dose should be calculated to neutralize the average dose of venom injected. This ensures that the majority of victims should be covered by the initial dose and keeps the cost of ASV to acceptable levels. The range of venom injected is 5 mg – 147 mg.

This suggests that the total required dose will be between 10 vials to 25 vials as each vial neutralizes 6mg of Russell's viper venom. Not all victims will require 10 vials as some may be injected with less than 63 mg. not all victims will require 25 vials. However, starting with 10 vials ensures that there is sufficient neutralizing power to neutralize the average amount of venom injected and during the next 12 hours to neutralize any remaining free flowing venom.

There is no evidence that shows that low dose strategies have any validity in India[41,49,53]. These studies have serious methodological flaws: the randomization is not proper, the allocation sequence was not concealed, the evaluators were not blinded to the outcome; there was no a priori sample size estimation, and the studies were underpowered to detect the principle outcome.

The same problem relates to high dosage regimen[42], often based on Harrison's textbook of medicine, which was written specifically for the U.S. snakes and not intended for use in the developing world.

No ASV test dose must be administered[43,44,45]

Test doses have been shown to have no predictive value in detecting anaphylactoid or late serum reactions and should not be used[27]. These reactions are not IgE mediated but complement activated. They may also pre-sensitize the patient and thereby create greater risk.

Recommendation for ASV administration

Neurotoxic/anti-haemostatic	8-10 vials initial
Repeat dosage in neurotoxicity	10 vials repeated in 2 hours if worsening of the clinical status and there is no improvement in clinical status.

Repeat dosage in haemotoxicity	5-10 vials are repeated if the clotting time is not normalized in 6 hours or if bleeding persists.

If the clinical condition worsens then it is managed only by supportive measures. No additional dosage of ASV should be given.

All ASV to be administered over 1 hour at constant speed.

Prevention of ASV reactions-Prophylactic Regimes

There is no statistical, trial evidence of efficient statistical power to show that prophylactic regimes are effective in the prevention of ASV Reactions.

The study by Wan Fan et al showed no benefit and the other two showed modest benefit[44,45]. However, because these studies were underpowered to detect the true outcome effect, larger clinical trials are needed to conclude that the prophylactic treatment is beneficial.

Two regimens are normally recommended

- 100 mg of hydrocortisone and 10 mg of H_1 antihistamine IV 1-2 minutes before ASV administration

- The dose for children is 0.2 mg/kg of antihistamine IV and 2mg/kg of hydrocortisone IV.
- 0.25-0.3 mg adrenalin 1:1000 given subcutaneously.

The conclusion in respect of prophylactic regimens to prevent anaphylactic reactions is that there is no evidence form good quality randomized clinical trials to support their routine use. If they are used then the decision must rest on the grounds, such as political policy in the case of government hospitals, which may opt for a maximum safety policy, irrespective of the lack of definitive trial evidence.

If the patient has a known sensitivity to ASV then premedication with adrenalin, hydrocortisone and anti-histamine is necessary.

ASV reactions

Anaphylaxis is life threatening, but despite the reluctance in giving ASV due to reactions, if the correct protocol is follow, it can be effectively treated and death with[46]. Anaphylaxis can be rapid onset and can deteriorate into a life-threatening emergency very rapidly. Adrenaline should always be immediately available.

The patient would be monitored closely and at the first sign of any of the following[47];

Utricaria, itching, fever, shaking chills, nausea, vomiting, diarrhea, abdominal cramps, tachycardia, hypotension, bronchospasm and angioedema

- ASV will be discontinued
- 0.5 mg of 1:1000 adrenalin will be given IM
- Children are given 0.01 mg/kg body weight of adrenalin IM
- 100 mg of hydrocortisone and 10 mg of H1 antihistamine, such as chlorpheniramine IV.

If in 10-15 min patient condition has not improved or is worsening, a second dose of 0.5 mg of adrenalin 1:1000 IM is given. This can be repeated for a third and final occasion. Hypotension and the hemodynamic instability should be managed accordingly with vasopressors.

Neostigmine in Snake bite

Neostigmine is an anticholinesterase that prolongs the life of acetylcholine and can therefore reverse respiratory failure and neurotoxic symptoms. It is particularly effective for post synaptic neurotoxins such as those of cobra and doubtful effectiveness for krait and Russells[48].

MATERIALS AND METHODS

This was a prospective analytical study. The study was conducted in a large teaching hospital and research institute catering population of forty to fifty lakhs. The period of study was 12 months. All the cases of definitive snake bite admitted in the toxicology centre were included as the subject of the study. Each case is only recorded as a definite snakebite if there is a clear description of a bite from a snake that was seen, or if there is clinical evidence of snakebite envenoming.

A detailed history regarding the type of work, the predisposition to snake bite, the preference to treatment (whether indigenous or modern), type of indigenous treatment underwent (leaves, powder, bark, root, others), the earliest symptom, the reason for coming to modern treatment, the accessibility to primary health care centre, the availability of the government or private doctor, the difficulty in transportation and the advice by the villagers was obtained. The varied clinical manifestation at presentation to the hospital was noted with particular interest to blood pressure, respiratory rate and effort, the conscious level, bleeding manifestations, local reactions and other vitals were documented.

The basic investigations viz., the routine blood investigations(complete blood count , blood urea, serum creatinine, blood sugar) was done along with liver function test and urine routine. Chest-X-Ray, USG abdomen was done as and when needed. The 20 minutes whole blood clotting time is done and patients with >20 minutes of clotting time was considered as abnormal coagulation.

Patient with ptosis, dysphonia, dysphagia, breathing difficulty, unconsciousness and opthalmoplegia were considered as neurotoxic. Bite site edema crossing the major joint is considered as criteria for local envenomation. Patient staying in the hospital for more than three days are considered for prolonged stay. These information's were analyzed on the basis of sociocultural, religious and regional beliefs, opinion towards indigenous medications, reasons for delayed reporting to modern medical care, sequence of symptoms and complications, course during the hospital stay, clinical outcome and socioeconomic implications. These cases were also were followed up for long term effect of snakebite on patients social and economic aspects. Respiratory failure, renal failure and prolonged hospital stay were considered as signs of serious envenomation and the significance between age, occupation, sex, bite to ASV timing and severity of the outcome were analyzed.

Patients with persistent bleeding tendencies were managed with blood products, renal failure patients were given dialysis support and respiratory failure is supported with ventilator. Relations between the timing of snakebite and the outcome in terms of renal failure, respiratory failure and prolonged hospital stay were analyzed. Whenever there were severe local reactions of the involved limb surgical management was instituted. Vascular surgical investigations and interventions were made on requirement basis. Doppler examination of the veins of the limb was done and appropriate surgical therapy like fasciotomy was instituted for the compartmental syndrome.

The study was approved by the institute ethics committee and informed consent was obtained from each patient. Continuous variables were expressed as mean +- standard deviation and discrete variable as percentage. The chi square test was used to examine the significance of difference.

RESULTS

Hundred of patients of snake bite became eligible for the study over a period of 12 months. Out of these 69 (69%) were male and the remaining females. The youngest patient was 11 years old. Usually the pediatric patients were referred to the Institute of child health situated 4 km from our hospital. The eldest was 75 yrs of age. In the present study 72% of the patients belonged to age group 10-40 yrs of age. Only 2% of the patient was above 70 yrs of age.

In the present study, most of the victims were agricultural workers. About 76% of the persons ligature as the first aid measure during the transport to the medical facility. Forty percent of the patients approached indigenous medical practitioner before coming to modern treatment. Out of the remaining 60%of the patient 31% approached government hospital and 29% of the patient approached private hospital.

The mean hours from the bite to time of ASV, administration was 14 hours. Ptosis was the commonest earliest manifestation of the victims. First symptom of toxicity was ptosis in 28% of the patients, gum bleed in 11% of the study population and double vision in 8% of the study population. The mean time of appearance of ptosis was 2 hours (range 1-21hours), gum bleed was 3 hours (range 1-6 hours),

double vision was 1 hour (range 1-4 hours), prolonged clotting was 2 hours (range 1-9 hours) and vomiting was 1 hour (range 1-3 hours). In the present study 34% were bitten by Russell's viper, 12% by saw scaled viper, 19% by cobra, 10% by common krait and 25% by non poisonous snakes.

Most of the bites were outdoor with only 9% occurring indoor. There were instances of snake bite during chores of household activities, playing, open air defecation and sleeping in the floor. The study population belonged to a perimeter of 50 to 75 km around Chennai. However, some patients were referred from for of places like Thiruvanamalai, Trichy and Madurai. In all the indoor incidences krait was the offending snake. Three percentage of the patient had multiple bites.

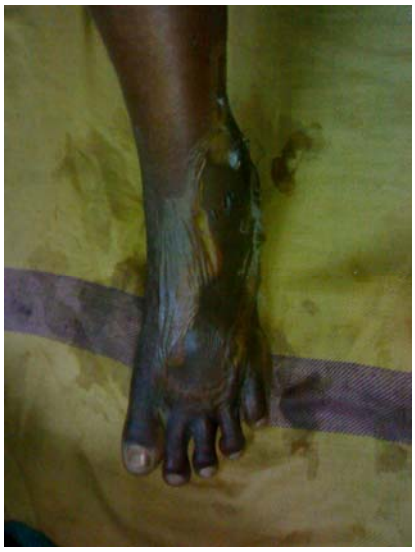
Regional lymphadenopathy is present in only 34% of the snake bites. Among these cellulitis and lymphadenopathy is seen commonly in Viperidae bite and cobra, krait causing insignificant local findings. Twenty eight of the study population went for complication. Out of which 13% of them were due to renal failure, 9% of the patient was due to respiratory failure necessitating ventilatory support. 10% of the study population needed supplementation with blood products. Seven percentage of the study population succumbed to snake bite, 85% of

which was due to ARF. The average period of study was 3-4 days. Persons with history of bleeding diathesis, neuromuscular problems, renal disease and unknown bite were not included in the study.

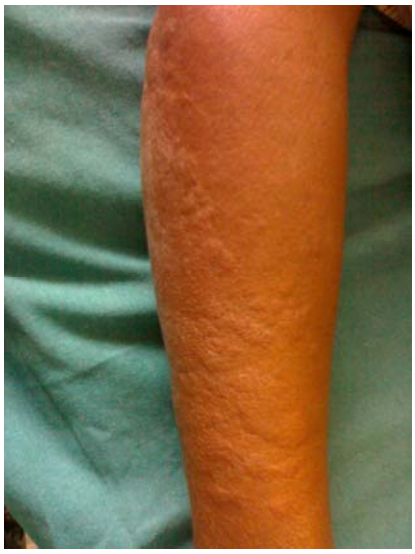
The various variables like sex, occupation, age, type of snake, site of bite, type of toxicity, time of first appearance of symptom, time of first dose of ASV and complications like ventilator support, dialysis, blood products, and death were compared. There was no significant relationship between the variables with ventilatory support.

Acute renal failure and dialysis requirement had definite correlation with some of the variables. Eighteen percent of the male patients developed renal failure which is significant at 5%. Agricultural workers had more predisposition to develop renal failure (27%).combined toxicity developed more frequent renal failure (36.8%). Time of first dose of ASV and need for hemodialysis had a significant correlation. Later the presentation to the health care facility, high incidence of renal failure requiring dialysis support. None of the patient who received ASV within 6 hours of bite went for renal failure. In contrast 26% of the patient who received ASV after 6 hours went in for renal failure and is highly significant at 5% level of significance.

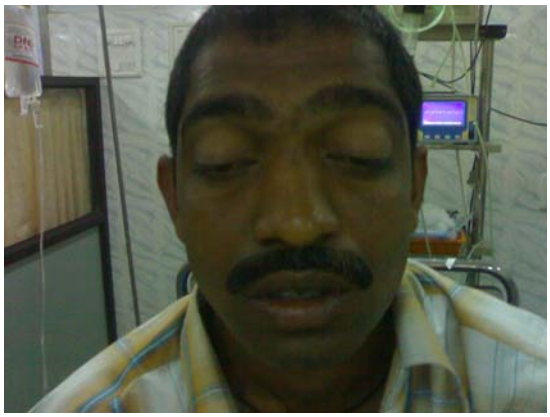
About 68.4% of the patients of combined toxicity had prolonged stay. Forty percentage of patients who received ASV after 6 hours had prolonged stay but only 22.9% of the patient who received ASV less than 6 hours had prolonged stay. All the patients who received ASV less than 6 hours are discharged. Fourteen percent of the patient who received ASV 6hours later died. Poisonous snake bite had elevated serum alkaline phosphatase. About 62.7% of the patient with poisonous snake bite had elevated SAP but only 16% of the non poisonous bite had elevated SAP.



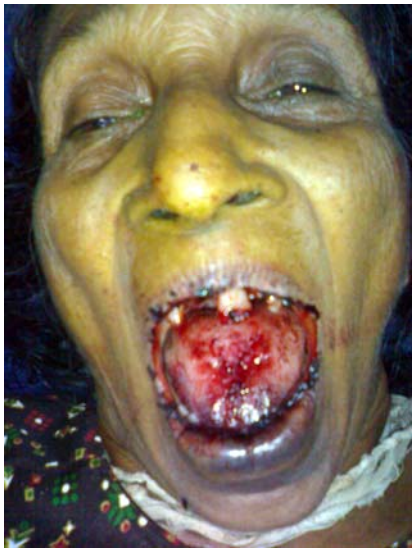
Cellulitis



ASV induced urticaria



Ptosis



Gum bleed

Hematuria

DISCUSSION

GENDER

In the present study, 69 persons were male. This male preponderance was similar to findings of other workers. Srimannarayana et al found that males are bitten more common[49]. Hansdac SG et al had found that snake bite were 2.5 times more common in males[50]. Meyer WP et al found 85% of the patients to be males, while the study carried out by Bhat RN in Jammu way back in 1974,70% of the study population wee males[51,52]. In the study by Paul V et al, male preponderance was 75%[53].

The high incidence of snake bite in the male is due to the following reasons. India is basically agricultural nation with rich tropical climate with lush vegetation. Males are more exposed to snake bite because they are the main breadwinners in the Indian family set up and women are housewife mainly in the rural setup. So this preponderance is mainly an occupational risk. In the case of urban population, the male children and adolescents are more commonly affected as they are more adventurous and explorative than female counterparts. In the rural setup, the female children and adolescents are mainly burdened with taking care of the family chores and males are expected to earn.

AGE

In the present study, more than 70% of the victims were within the age group 11-40 years. Only 2% of the victims were more than 70 years old. This was similar to the observation made by Thomas G Glass who found 74% incidence in the age of 10-70 years while only 2% were more than 70 years[54]. In the study by Narvencar K 60% of cases belonged to 11-40 years of age similar to the present study[55].

The youngest patient was 11 years of age and the oldest was 75 yrs of gage. The youngest got bitten while going for open air defecation and the oldest got bitten while attending work. Due to poor socioeconomic condition, the elderly are compelled to work and the poor eyesight and the poor general condition makes them an easy target for the provoked bites. This was similar to the study by Narvencar K where the youngest patient was 12 yrs and the oldest was 70 yrs old[55].

COMMONLY ENCOUNTERED SPECIES

In the present study 39% were due to Russell's viper, 19% were due to cobra and 12% was due to saw scaled viper, 10% were due to krait and 25% were due to non poisonous bites.

BITE TO NEEDLE (ASV) TIME

The average time delay between bite and ASV administration was found to be 14 hours. This was similar to many other studies in India. In the study by Srimannarayana et al, the average time was 18.3 hours[49]. The delay was due to various reasons and there is significant relation between the delay of ASV administration and outcome.

The delay in ASV administration was due to the following reasons

- Consulting the indigenous medical practioner first and then coming for modern medicine consultation.
- Abiding by local customary beliefs and performing pooja's at local temple.
- Lack of transport facilities at some of the remote villages and the cost of the transport to the tertiary care centre.
- Fear for the cost of treatment at the modern health care system.
- Availability of the indigenous medicine persons at proximity
- Inadequate experience of the primary health care physician[56].
- Hesitation by the Primary Health Officer to institute ASV for the fear of anaphylactic reactions

TREATMENT PREFERENCE

Forty percentage of the patient went in for indigenous medicine first before attending modern health care facility/ in India, which is basically a Hindu country, various age old religious beliefs and customs, people tend to think that snake bite is due to previous karma and indulge in various offers and pooja's. India is also the birthplace of Ayurveda, a branch of complementary medicine. Though there are proven good methods are available for the treatment of various ailment, the country is steaming with many quacks who offer inappropriate treatment, most commonly leading onto various complications. Thus, the people's preference to indigenous medicine is due to the following reasons

- Easy availability of the indigenous medical practioner in rural areas. As most of the people hail from rural area and the indigenous medical practioner lives in the community itself the people tend to consult him first causing unnecessary delay.
- Victims, as advised by the elderly seek indigenous medical practioner.
- As 70%-80% of the bites are nonpoisonous and these poisonous bites are claimed to be successfully treated by the indigenous

medical practioner, people tend to consult him for poisonous bite treatment also.

- Custom and religious beliefs compel them to go for other unproven modalities of treatment like performing pooja's and offerings.
- Indigenous medicine is relatively cheaper than modern medicine.
- Most of the people believe that the indigenous medicine would halt the progress of envenomation, buying them adequate time to reach modern health care.
- False propaganda by the indigenous medical practioner that they can cure all kind of envenomation.
- Unavailability of transport facility at remote areas.

LIGATURE AS FIRST AID

In the present study 76% of the victims resorted to tying tight ligature proximal to the bite site. The victims had a strong belief that the ligature prevented any advancement of the venom into the systemic circulation. The various scenes in the entertainment media like TV and cinema also reinforced this false belief. Some of the victims also related their survival to their act of tying the ligature immediately above the site

of snake bite. The victim's behavior is also reinforced by elder's beliefs and opinions.

COMMONEST SYMPTOM

In the present study ptosis was the commonest symptom encountered (28%). Ptosis was the presenting features in most of the elapid envenomation and some of the Viperidae envenomation also especially Russell's viper bites. The median time of appearance of ptosis was 2 hours with range of 1-21 hours. Neurological symptoms occurred early in cobra bite and Russell's viper bite. The neurological manifestation in krait was delayed, sometimes delayed by 21 hours. This might probably due to slow absorption of the venom being deposited in the skin deep or subcutaneous tissue by short and sharp fangs. This delayed onset of symptoms naturally lead to indifference towards the seriousness of the bite and hence delayed reporting to the hospital.

Next most common symptom was gum bleed noted in 11% of the study group. This in contrast to the Srimannarayana study where hemetamesis and hematuria were the commonest presenting complaint, (37.7%) and (34.4%) respectively[49].

Vomiting was also one of the commonest symptoms, around 10% of the victims presented with it. Most of the episodes of vomiting

occurred after consuming indigenous medicines (powder and leaves), and it is difficult to conclude whether the vomiting is due to systemic envenomation or due to indigenous medication itself. In 8% of the study, population double vision was the earliest manifestation.

The symptoms like gum bleed, ptosis and persistent vomiting, compelled the victim to come for modern medicine after consulting the indigenous medical practioner. In few cases, hematuria and hemetamesis were the earliest manifestation.

POPULATION AT RISK

In the present study, the commonly affected group was agricultural laborers and manual laborers. The subjects of the study were from 50-75 km around Chennai, mainly involved in paddy and sugarcane cultivation. This finding confirms that snake bite is occupational hazard. In the urban and semi urban population people involved in carpentry and those involved in clearing of bushes for building construction purposes were mainly affected.

OUTDOOR VERSUS INDOOR

In the study 91% of the bites took place in the outdoor activity. This proves that snake bite occurs when people interferes in snake’s

habitat rather than the vice versa. There were quite a few instances where snake bite happened in the indoor activities (9%). In all these instances common krait was the offending snake. This is in accordance with earlier findings by various authors where people sleeping on the floor got bitten by krait. The significance of this is that most of the time people couldn't confirm that it was snake bite and there is always a possibility to ignore it as insect bite, as coincidently krait bite doesn't cause any local cellulitis. This is one of the determining factors in the late presentation to health care facility. In a recent incident in suburban Chennai, three children of the same family succumbed to krait bite while they wee sleeping on the floor. This is due to the habit of the snake entering the house in search of prey like rats and other rodents.

SITE OF BITE

In the present study, 83% of the snake bite site was lower extremity. The remaining 17% of the cases involved upper extremity. The study also confirmed that most of the cases occurred during barefoot walking. Rural Indians are not used to protective wear like shoes, especially working in the field.

SINGLE OR MULTIPLE BITE

Ninety seven percentage of the bite was single, confirming that snake bite occurs as a defensive act by the snakes. Only in three cases, there were multiple bites. In all those cases, the snakes are handled and no way of escape for the snake, so it repeatedly attacked the victim.

LOCAL ENVENOMAION

In the present study 63% of the patient developed edema and cellulitis at the bite site. Viper and cobra bite are the most common offending species. In the case of krait bite the local envenomation is minimal or sometimes absent. This is similar to many studies in the past suggesting the absence of local envenomation by the krait bite.

REGIONAL LYMPHADENOPATHY

In the present study 34% of the patient developed regional lymphadenopathy. Most of the bites were due to viper and cobra. The incidence of regional lymphadenopathy in krait bite is very minimal.

RENAL FAILURE

In the present study, 11% of the patients had acute renal failure requiring dialysis therapy. In India the incidence of ARF following snake bite varied from 13 to 32%[57,58,59]

a.. In the study by Paul V et al, the incidence of renal failure is 26% and in study by Srimannarayana J 50% of the patients went in for renal failure[49,53]. In Srimannarayana study, only hemotoxic snake envenomation was considered for study[49]. That could have been the reason for disparity in the percentage of renal failure.

The correlation between time of administration of ASV and the propensity for renal failure was studied. None of the patient who received ASV within 6 hours went for renal failure. But 26% of the patient who received ASV 6 hours later went in for renal failure. This was found to be significant at 5% level of significance. This confirms that early administration of ASV is associated with decreased incidence of renal failure. This is similar to outcome in various other studies. In the study by Paul V et al 35.7% of the patient who arrived within six hours of bite developed ARF, while the incidence of ARF among the patient who arrived after 24 hours was 80.6%(P value <0.001)[53].

ASV neutralizes the circulating venom only and no amount of ASV will neutralize or combine with venom once the venom is attached or adsorbed to target organ i.e. platelets, RBC, vascular endothelium, renal tubules, muscle and neuromuscular receptors. So whenever ASV is administered lately, the renal damage had already occurred and the ASV cannot prevent the renal failure.

These findings are similar to the observations made by Vijeth SR that the incidence of complications was directly proportional to duration of venom in the blood prior to neutralization by ASV and Ash T et al that there is positive correlation between severity of renal failure and increased interval between bite and ASV administration.

VENTILATOR SUPPORT

In the present study about 9% of the patient went in for respiratory failure requiring ventilator support. In the study by Paul V et al, the incidence of ventilator support was 6%. There was no positive correlation between the delayed administration of ASV and higher incidence of respiratory failure requiring ventilator support.

The onset of neurological symptoms, culminating in respiratory failure, depends upon the site of bite, amount of venom injected and the post bite exertion or exercise by the victim

DEATH

The mortality of the present study was 7%. Among that, 85% was due to renal failure and the remaining was due to neurological complication. In the study by Paul V et al, the mortality was 14% in the high ASV dose group and 10% in the low ASV dose group[53]. In the

study by Srimannarayana, the morality rate was 22.2%, 85% of which is due to ARF similar to the present study[49]. None of the patient who received ASV within six hours died but 14% of the patients who received ASV six hours later died. This was significant at 5% level of significance. All these observations reinforce the importance of early administration of adequate ASV and prevention of morbidity and mortality.

AVERAGE HOSPITAL STAY

Average stay period of stay at toxicology ward was 3.71 days. This is in contrast to the finding by Paul V et al, where in average period of hospital stay was 9.02 in the high dose group and 8.42 in the low dose group[53]. The difference in finding may be due to the fact that the present study takes into consideration only the stay at the toxicology ward. Once the patients are stabilized, they were shifted to general ward for further observation and treatment.

CONCLUSION

This study on snake bite has come out with various conclusions, some of them supporting the earlier studies and some of them contradicting the previous ones.

The male preponderance and the highest number of victims in the productive age group stamps snake bite as an occupational hazard and urges to make implementation of preventive measures. The incidence of snake bite can be reduced by the following methods,

1. Providing protective gear (shoes and gloves) for the agricultural workers.
2. Advising people to carry torch while going to fields during night.
3. Avoid exploring the dense vegetation and fields during the night hours
4. Advising them to carry a stick or cane and to tap the pathway to create vibrations. Snakes actually move out of the pathway sensing the vibration

The delay in the arrival to the hospital and the harmful effects of nonscientific methods can be reduced by the following;

1. Regulating the media and the press in spreading of unscientific methods of treatment.
2. Sensitizing the people regarding the dangers of adopting unscientific methods.
3. Educating the people regarding the presence of Antisnake venom and it's almost 100% efficiency in preventing death.
4. Educating people regarding the dangers of ligature.
5. Training the community health worker in management of snake bite victim in the first aid and early hospital referral.
6. Regulate the indigenous medical practitioners and licensing them properly.
7. Reinforce the primary health care centers and district hospitals to treat the snake bite victims and serve as a proper chain of referral.
8. Training the medical officers in primary health care centre in the management of snake bite.

The delay in the administration of ASV can be prevented by properly training the primary health centre doctor in the rational use of ASV. Though ASV is administered in the primary health centre adequate dose is not given leading to prolonged period of free circulating venom and subsequent complications. Taking all these into

consideration the national snake bite task force was formed and national treatment protocol for snake bite have been modulated and in the process of implementation throughout the country. With this multipronged approach the mortality and morbidity associated with snake bite can be drastically brought down.

BIBLIOGRAPHY

1. White J. Bites and stings from venomous animals: a global overview. Ther Drug Monit 2000; 22; 65-8.

2. Warrell DA. The clinical management of snake bites in the Southeast Asian Region. Southeast Asian J Tropical Med Public Health 1999; 30:1-84

3. J Srimannarayana et al., Rational Use of Anti-snake Venom, JIPMER, JAPI

4. Narvencar K. Correlation between timing of ASV administration and complications in snake bite.

5. Dutta TK, Ghotekar LH. Rational use of anti snake venom. Apicon medicine update.

6. Minton S A. is there any non venomous snakes? An update on colubrid envenoming. Adv herpetoculture 1996; 127-134

7. Knight A, Densmore L D & Rael E D. Molecular systematics of the Agkistrodon complex. In Campbell J A & Brodie E D (eds).Biology of the pit vipers. Tyler, TX: Selva, 1992:49-69

8. Kochva E. Oral glands of the reptilian. In Gans C & Gans K A (eds) Biology of reptelia. London: Academic Press, 1978:43-163

9. Harvey A L (ed.). Snake toxins. International Encyclopedia of Pharmacology and Therapeutics, Section 134. New York: Pergamon, 1991

10. Meier J & White J (eds). Handbook of clinical Toxicology of Animal Venoms and poisons. Boca Raton, FL: CRC Press, 1995.

11. Douglas W W.Polypeptides: Angiotensin, plasma kinins and others. In Goodman A G , Gilman L S, Rall T W & Murad F (eds) The Pharmacological Basis of Therapeutics.

12. Hutton R A & Warrell D A. Action of Snake venom components on Hemostatic System. Blood Rev 1993; 7;176-189.

13. Myint-Lwin, Warrell D A, Philips R E, Tin-Nu-Swe. Bites by Russell's Viper in Burma:Hemostatic, Vascular and renal disturbances and response to treatment. Lancet 1985; ii: 1259-1264.

14. Ratcliff P J, Pukrittayakamee S, Ledingham J G G & Warrell D A. Direct Nephrotoxicity of the Russell's viper venom

demonstrated in the isolated perfused rat kidney. Am J ATrop Med Hyg 1989; 40:312-319.

15. Sitprija V & Chiyabhut N. Nephrotoxicity in snake envenomation. J Nat Toxins 1999; 8:271-277.

16. Warrell D A. Sea snakes bites in the Asia Pacific region. In Gopalakrishnakone P (ed.) Sea Snake Toxicology. Singapore University Press, 1994: 1-36.

17. Warrell D A & Ormerod L D.Snake venom opthalmia and blindness caused by Spitting Cobra in Nigeria. Am J Trop Med Hyg 1976; 25:525-529.

18. Tin-Myint & Warrell D A. bites by king cobra in Myanmar: Successful treatment of severe neurotoxic envenoming. Q J Med 1991: 80:751-762.

19. Bicher HI, Roth M, Gitter S. Neurotoxic activity of Vipera palestenia venom. Depression of central autonomic vasoregulatory mechanisms. Med Pharmacol Exp.1966;14:518-28.

20. Chugh KS. Snake venom induced renal failure in India. Kidney Int1989;35:891-907

21. Khan MJ, Hemker HC. Studies on blood coagulation factor V. Changes of molecular weight accompanying activation of factor V by thrombin and the procoagulants protein of Russells viper venom. Thromb Diath Haemorrh 1972;29:25-3222.

22. Kisiel W, Hermodson MA, Davie EW. Factor X activating enzyme from Russells viper venom: isolation and characterization. Biochemistry 1976;15:4901-6.

23. Aung W, Kyaw KP, Hla B et al. Renal involvement in Russells viper bite paients without disseminated intravascular coagulation. Trans R Soc Trop Med Hyg 1998;92:322-4.

24. Sant SM, Purandare NM. Autopsy study of case of snake bite with special reference to Renal lesions. J Postgrad Med 1972;18:181-8.

25. Gundappa RK, Sud K, Kholi HS, et al. Snake bite induced acute interstitial nephritis : report of a rare entity. Renal Failure 2002;24:369-72.

26. Banerjee RN. Poisonous snakes of India, their venoms, symptomatoloty and treatment of envenomation. Progress in

clinical medicine in India 2nd seires. Ed Ahuja MMS. Arnold Heinmann. Delhi 1978:136-78.

27. Warrell D A. Environmental and Genetic Disorders. Animal Toxins. 32:581-618.

28. Chugh KS, Paul Y et al.Acute renal failure following snake bite. Am J Kidney Disease 1984;4:30-8.

29. Peiris OA et al. Exchange transfusion in the treatment of Russells viper bite. Postgrad Med J 1969;45:625-9.

30. Date A,Pullimood R et al. Hemolytic uremic syndrome complicating snake bite. Nephron 1986;42:89-90.

31. Simpson ID Snake bite: Recent Advances 2006 in Medicine Update 2006 Ed Sahay BK. The Association of Physicians of India 639-643.

32. Draft Summary Protocols. Indian National Snakebite Conference.

33. Sharma et al. Impact of Snake bites and determinants of fatal outcome insoutheastern Nepal. Am J Trop Med Hyg. Aug;71:234-8.

34. Sutherland et al. Rationalisation for first aid methods for elapid snakebite. Lancet 1979;183-186.

35. Howarth DM et al. Lymphatic flow rate and first aid in simulated peripheral snake or spider envenomation. Medical Journal of Australia 1994;161:695-700.

36. Reid HA et al. Prolonged coagulation effect in Malayan Pit Viper bite. Lancet 1963;1:621-26.

37. Greenwood B, Warrell DA, et al. Immunodiognosis of snake bite. BMJ 1974;743-45

38. Theakston RD et al. Micro-Elisa for detecting and analyzing snake venom and antibody. Lancet 1977(2)639-41.

39. Ho M et al. A Critical reapprisal of use of enzyme linked immunosorbant assay in the study of snake bite. Toxicon 1986;24:211-221.

40. Tun P et al. Amount of venom injected by Russells viper. Toxicon 1986;24(7):730-733.

41. Agarwal PN. Management of Respiratory failure in severe neuroparalytic snake envenomation 2001. Neurol India 49(1)25-28.

42. Wallace JF. Disorders caused by venoms, bites and stings. Harrisons principles of internal medicine. Vol II. 16th edition.

43. Wen Fan H, et al. Sequential randomized and double blind trial of promethazine prophylaxis against early anaphylactic reactions to anti venin for Bothrops snake bite. 1999 BMJ(318) 1451-1453.

44. Premawardenha A, De silva CE et al. Low dose subcutaneous adrenalin to prevent acute adverse reactions to antivenom serum in people bitten by snakes, randomized placebo controlled trial. BMJ 318;1041-43.

45. Gawarammana et al. Parallel infusions of hydrocortisone without or without chlorpheniramine bolus injection to prevent acute adverse reactions to antivenom for snakebites. Med J Aust 2003; 180:20-23.

46. Kalantri S et al. Clinical predictors of in-hospital mortality in patients with snake bite. A retrospective study from a rural

hospital in ventral India 2005. Tropical Medicine and International health 11(1) 22=30.

47. Peshin SS, Lal SB et al. Snake envenomation in Ed la. Snake bite management of common India. Snake and insect Bite 1997 National Poison information centre AIIMS Delhi.

48. Watt G, Padre L, et al. Torniquet application after cobra bite. Delay in onset of neurotoxicity and danger of sudden release. 1988 AM J Trop Med Hyg 87;618-622.

49. Srimannarayana J et al. Rational use of Anti-Snake Venom(ASV). Trial of various regimens in Hemotoxic snake envenomation. JAPI 2004;788-93.

50. Hansdak SG, Lallar KS. A Clinicoepidemiological Study of snake bite in Nepal. Tropical Doctor 1998;28:233-6.

51. Meyer WP, et al. First clinical experience with a new ovine FAB Echis ocillatus snake bite antivenom in Nigerial. Am J Trop Med Hyg 1997;56:291-300.

52. Bhat RN. Viperine snake bite poisoning in Jammu. J Indian Medcial Assoc 1974;63:383-92.

53. Paul V,et al. High dose anti-snake venom versus low dose anti-snake venom in the treatment of poisonous snake bites-a critical study. J Assoc Phy. India 2004;52:14-17.

54. Thomas G Glass. Early debridement in pit viper bites. JAMA 1976;235:2513-16.

55. Narvencar K. Correlation between timing of ASV administration and complication in snake bite. JAPI sep 2006;54:717-19.

56. Bawskar HS and Bawaskar PH. Call for global snake bite control and procurement funding. Lancet 2001;357:1132-33.

57. Shastry JC, Date A, Carman RH, Jonny KV. Renal Failure following snake bite. Am J of Trop Med Hyg 1977;26:1032-8.

58. Mittal BV, Kinnare SG, Acharya VN. Renal lesions following viper bites-A study of 14 years. Ind Jour Of Med Res 1986;83:642-51.

59. Mathew MT et al. Renal papillary necrosis in ophitoximia. J Assoc Physician India 1987;35:20.

PROFORMA

Snake Bite Case Sheet (Tick appropriate)

Name : Age :

Sex : (if Female date of last menstrual period)

Antenatal Y/N Lactating Y/N

IP No. Toxicology No. :

Address :

Rural / Urban / Semi-Urban

Informant : Reliability : Good/ Fair / Poor

Snake Bite (definite / alleged / unknown)

Snake Bite details :

Snake type (if brought by Attenders)

Location of the Incident :

Indoor (home) / Outdoor (work place etc.)

Site of bite : Time of Bite : Single Bite / Multiple Bites / Serial Bite :

Patients position at the time of Bite : Sleeping / Working/Bare foot-Y/N

First aid method used :

Refusal of first aid Y/N :

Synopsis of Treatment given Outside :

First aid given by Physician/ Non physician Medical Person / Non Health Care person :

TT given Y/N

H/O Allergy, Asthma, Atopy, Previous ASV administration :

Other Medical Illness (details) :

Clinical features

Vital signs :

Pulse :

BP (Lying) (Sitting) (Standing)

Respiratory Rate : Depth (shallow / deep / normal) :

JVP

General symptoms :

Nausea, vomiting, postural giddiness, Abdominal Pain, Drowsiness

Local symptoms / Signs :

Pain Y/N, Warmth Y/N, Fang Marks Y/N.

Extent of Swelling (in relation to joints)

Skin Colour changes Y/N, Bisters Y/N, Parasthesia Y/N, Hypoaesthesia Y/N :

Regional Lymphadenopathy Y/N :

Distal pulses Y/N, capillary filling time normal / abnormal, Distal sensation Y/N :

Coagulopathy

Epistaxis Y/N, Gum Bleed Y/N, Hematemesis / Malena / Hematochezia Y/N, Hemoptysis Y/N

High Colur urine Y/N, Vaginal bleed Y/N

Headache / seizure Y/N

Skin Petechiae, Purpura, Y/N

Oral mucoal bleeds / gingival bleed Y/N, conjuctival bleed Y/N

Bleed from the site of bite / recent wounds Y/N :

Neurotoxic :

Sense of Smell : Altered : Y/N (if Yes, give details)

Sense of Taste : Altered : Y/N (if Yes, give details)

Drooping of eyelids Y/N :

Diploma Y/N, Dysphonia Y/N, Dysphagia Y/N, Nasal regurgitation Y/N :

Shortness of breath Y/N :

Weakness of limbs : (upper limb power /5, lower limb power /5)

Glasgow coma scale :

Pupils

Extraocular movement (full / Restricted)

Facial muscles (unable to hold air in mouth Y/N)

Gag reflex / palatal movement (normal / abnormal)

Pooling of secretions oral cavity Y/N :

Spontaneous breath count :

Meningeal signs Y/N :

Fundus :

Cardiovascular :

Giddiness / Faintness Y/N, frothing at mouth Y/N :

Renal :

Loin pain/tenderness Y/N, High coloured urine Y/N, Oligoanuria Y/N, hiccups Y/N, Pleuritic chest pain Y/N :

Musculotoxic :

General Myalgia Y/N, Pain, Stiffness Y/N, tenderness of muscles Y/N, Trismus Y/N.

CVS/RS/Abdomen Examination

Investigations

Complete blood count (include platelets, peripheral smear study) ;

Urine gross colour and microscopy (on admission) (After 8 hours)

20 Minute whole blood clotting time (note for serum colour)

Liver function tests :

SB/DB :

ALT/AST/ALP

CPK (Optional)

PT, Aptt :

ABG (Optional; May be risky in coagulopathy)

ECG

SYNDROME CYTOTOXIC / HEMOTOXIC / NEUROTOXIC / COMBINATION (SPECIFY)

Sr No	Dt of Admission	Time	Name	Age	Sex	IP No	Place	In/Outdoor	Work	Occupation	Site of Bite UL	Site of Bite LL	Time	No of Bite	ligature
1	22.08	12 50 am	Rangasamy	60	M	57015	Tiruvllur	Outdoor	Arranging things after work	Carpenter		Lt Dorsum Foot	5pm	Single	Y
2	24.08	10 00 am	Shankar	34	M	57546	Poompuhar/Villivakkam	Outdoor	Saw snake entering into arranged things	Building coolie	lt middile finger		1030pm	Single	Y
3	21.08	90 00 pm	Vijayalakshmi	34	F	57525	Sriperampathur	Outdoor	Taking woods for make fire	Coolie		Rt Dorsum Foot	5pm	Single	Y
4	27.08	03 20 pm	Mani	52	M	58386	Hundai Company.Cipcot	Outdoor	Taking gunny bag in fields	Cook	lt thump		09 15 am	Single	Y
5	22.08	12 noon	Kanniyammal	35	F	57210	Chengalpattu	Outdoor	Cutting wood	Coolie		Rt Dorsum Foot	0800am	Single	Y
6	16.08	08 00 am	Velu	55	M	47334	Madurai	Outdoor	Cutting wood in the forest	Coolie		Rt Dorsum Foot	10 00 am	Single	Y
7	2.07	08 00 am	Ramesh	30	M	50186	V.Kotta Andra	Outdoor	Mango farm	Agriculture		Lt Dorsum Foot	03 30 pm	Multiple	Y
8	15.06	09 00 am	Thimmaiah	35	M	39858	Andra Pradesh	Outdoor	Sugrcane farm	Agriculture		Rt Dorsum Foot	10 50 am	Single	Y
9	16.03	0445 am	Annadurai	34	M	17238	Villupuram	Outdoor	Ground nut farm	Agriculture		Rt Dorsum Foot	0400am	Single	Y
10	22.03	0430 am	Ramalingam	63	M	18871	Cheyyur	Outdoor	Paddy field	Agriculture		Rt Dorsum Foot	0400am	Single	Y
11	27.02	12 50 am	Madavan	48	M	13154	Tiruvannmai	Outdoor	Ground nut farm	Agriculture		Lt Dorsum Foot	1100 am	Single	Y
12	25.02	0830pm	Kannaiyan	55	M	12826	Kerapakkam	Outdoor	Manuring the field	Agriculture		Lt Dorsum Foot	0500pm	Single	Y
13	7.03	0625pm	Subramani	65	M	15279	Arrakonam	Outdoor	Ground nut farm	Agriculture		Lt Dorsum Foot	0345am	Single	Y
14	23.02	0500pm	Ravi	20	M	12579	Tiruvanamalai	Ourdoor	Sugrcane farm	Agriculture		Rt Dorsum Foot	0900am	Single	Y
15	20.02	0715pm	Ramalakshmi	65	F	48802	Tiruvallur	Outdoor	Sugrcane farm	Agriculture		Suspected Over Vein	0300pm	Single	N
16	12.08	0700 pm	Usha	19	F	54540	Arakonam	Outdoor	Farm near home	Agriculture		Lt Leg Dorsal Aspect	0730pm	Single	Y
17	28.08	630am	Mahalingam	36	M	50857	Gumidipoondi	Outdoor	Around home	Agriculture		Lt Leg Third Toe	830pm	Single	N
18	25.07	1045pm	Manikandan	27	M	50187	Arakonam	Outdoor	Sugarcane farm	Agriculture		Rt Foot Dorsum	0300pm	Single	Y
19	5.07	130am	Ramaraj	50	M	44573	Thiruvallur	Outdoor	Fields working	Agriculture		Rt Foot Dorsum	0630pm	Single	Y
20	16.07	11pm	Kanniappan	50	M	47649	Sriperampathur	Outdoor	Open air toilet	Coolie		Rt Foot Dorsum	0800pm	Single	Y

Sr No	Dt of Admission	Time	Name	Age	Sex	IP No	Place	In/Outdoor	Work	Occupation	Site of Bite UL	Site of Bite LL	Time	No of Bite	ligature
21	19.07	1110pm	Vasantha	45	F	48523	Arokonam	Outdoor	Grass cuting	Agriculture		Rt Great Toe	0300pm	Single	N
22	16.07	0230pm	Devi	21	F	47588	Thiruvallur	Outdoor	In front of the house	House wife		Lt Great Toe	0700pm	Single	Y
23	27.01	1245pm	Shenbagavalli	37	F	5875	Thiruvallur	Indoor	Cooking	House wife		Lt Dorsum Foot	1100pm	Single	Y
24	30.03	1000am	Murugan	30	M	20670	Tiruvanamalai	Outdoor	Barefoot walking	Coolie		Lt Foot Dorsum	500pm	Single	Y
25	19.03	0930pm	Harikrishnan	20	M	18053	Mothampedu	Outdoor	Picking stone	Unemployed	lt index finger		0630pm	Single	Y
26	19.03	0215am	Baskar	34	M	17788	Gumidipoondi	Outdoor	Urinating in wayside	Agriculture		Lt Dorsum Foot	0800pm	Single	Y
27	16.03	0625am	Sheeba	19	F	17442	Arakonam	Outdoor	Cleaning house	Sudent	lt middle finger		0615am	Single	Y
28	11.03	1100pm	Elumalai	45	M	16103	Thiruvanamalai	Outdoor	Sugarcane farm	Agriculture		Rt Dorsum Foot	10 15 am	Single	Y
29	4.03	1235 am	Elumalai	50	M	14389	Gumidipoondi	Outdoor	Working in fields	Agriculture	lt little finger		0400pm	Single	Y
30	3.04	0230am	Asokan	30	M	2142	Thiruvanamalai	Outdoor	Keeping things in sugrcane field	Agriculture		Lt Foot Dorsum	0600pm	Single	Y
31	9.04	0400am	Muthu	13	M	22727	Neelangarai	Outdoor	Barefoot walking	Student		Rt Great Toe	1000pm	Single	Y
32	7.04	0700am	Vennila	41	F	22403	Thiruvallur	Outdoor	Barefoot walking	Coolie		Lt Dorsum Foot	0830pm	Single	Y
33	31.05	0830pm	Praveen	15	M	36010	Arokonam	Outdoor	Barefoot walking	Student		Rt Dorsum Foot	0800pm	Single	
34	1.06	1255am	Gunasundari	19	F	36042	Perambur Chennai	Outdoor	Around home,barefoot	Coolie		Lt Foot Dorsum	1200am	Single	Y
35	2.07	0900am	Villisamy	47	M	43687	Pondichery	Outdoor	Around home,barefoot walking	Coolie		Rt Foot Dorsum	1200am	Single	N
36	27.07	1230am	Manikandan	25	M	50487	Kanchipuram	Outdoor	Returning home,barefoot walking	Agriculture		Rt Foot Dorsum	0700pm	Single	Y
37	31.07	0730pm	Dhanam	40	F	51186	Sriperampathur	Outdoor	Around home,cleaning	Coolie	lt hand		0230pm	Single	Y
38	4.07	1030pm	Selvaraj	25	M	37003	Sriperampathur	Outdoor	Returning home,barefoot walking	Coolie		Lt Great Toe	0730pm	Single	Y
39	6.06	0830am	Chiranjeevi	25	M	37349	Gumdipoondi	Indoor	Sleeping	Agriculture	rt fore arm		1230am	Single	Y

Sr No	Dt of Admission	Time	Name	Age	Sex	IP No	Place	In/Outdoor	Work	Occupation	Site of Bite UL	Site of Bite LL	Time	No of Bite	ligature
40	3.07	0700am	Navaneetham	43	F	44011	Thiruvallur	Indoor	Sleeping	Coolie		Rt Great Toe	0400am	Single	N
41	16.06	0930am	Sumathy	25	F	39861	Thiruvallur	Outdoor	Around home,open air toilet	Student		Lt Dorsum Foot	1000pm	Single	Y
42	25.01	0100am	Natarajan	36	M	97107	Thiruvallur	Outdoor	Paddy field/opening water	Agriculture	rt index finger		0330pm	Single	Y
43	23.01	0200am	Kotteswaran	30	M	4752	Thiruvallur	Ourdoor	Barefoot walking	Agriculture		Rt Dorsum Foot	0700pm	Single	Y
44	3.02	1215pm	Thenpandian	11	M	7523	Kolathur	Outdoor	Open air toilet	Student		Gluteal Region	0600AM	Single	N
45	12.02	1145am	Subramani	23	M	9593	Thiruvallur	Outdoor	Barefoot walking	Agriculture		Rt Dorsum Foot	0400pm	Single	N
46	19.03	0900pm	Ramados	55	M	10848	Pooneri	Outdoor	Ground nut farm	Agriculture		Lt Dorsum Foot	0345pm	Single	Y
47	9.03	1030am	Das	42	M	15662	Kodingayur	Outdoor	Steel plant	Machine worker	rt thumb		0945am	Single	N
48	6.03	0430pm	Ethiraj	55	M	15025	Arokonam	Outdoor	Barefoot walking	Agriculture		Lt Dorsum Foot	0700pm	Single	N
49	15.03	1230am	Renuka	32	F	16950	Manali	Outdoor	Barefoot walking	Coolie		Lt Dorsum Foot	100pm	Single	N
50	13.04	0100am	Shanthi	23	F	23839	Andra Pradesh	Outdoor	Barefoot walking	Student		Rt Foot Dorsum	0730pm	Single	Y
51	15.04	0800am	Munian	35	M	24294	Kanchipuram	Outdoor	Paddy field	Agriculture		Rt Foot Dorsum	0730pm	Single	N
52	3.06	0430pm	Poongavanam	32	F	36594	Tiruvnamalai	Outdoor	Barefoot walking	Agriculture		Rt Foot Dorsum	0710pm	Single	N
53	5.07	0630pm	Bhavani	17	F	44836	Tiruvanamalai	Indoor	Arranging things after	Student		Rt Leg	0700pm	Single	Y
54	6.08	11.30am	Mhadavan	29	M	52999	Thiruvalur	Outdoor	Brinjal field	Agriculture		Rt Thigh	0600am	Single	N
55	5.08	1040pm	Bhavani	12	F	52838	Chennai	Outdoor	Barefoot walking	Student		Lt Foot Dorsum	0730pm	Single	N
56	27.07	900pm	Stephen	21	M	50759	Sholinganalor	Outdoor	Bathing	Student		Rt Foot Dorsum	0730pm	Single	N
57	18.06	1230am	Reshmi	30	F	40139	Perungudi	Outdoor	Barefoot walking	House wife		Lt Foot Dorsum	1100pm	Single	N
58	15.06	1030pm	Dhanasekar	30	M	39829	Avadi	Outdoor	Barefoot walking	Government emplo		Lt Foot Dorsum	0800pm	Single	N

Sr No	Dt of Admission	Time	Name	Age	Sex	IP No	Place	In/Outdoor	Work	Occupation	Site of Bite UL	Site of Bite LL	Time	No of Bite	ligature
59	2.07	0930pm	Karthik	28	M	43973	Porur	Indoor	Arranging tings	Coolie		Lt Great Toe	0530pm	Single	Y
60	16.07	0500pm	Anu	22	M	47608	Avadi	Indoor	Sleeping	House wife	lt hand elbow		0330pm	Single	N
61	24.07	0320am	Latha	25	F	49634	Madarambakam	Indoor	Sleeping	House wife		Rt Foot Dorsum	1230am	Single	Y
62	31.01	0815am	Ravi	30	M	6665	Ponneri	Outdoor	Barefoot walking	Government emplo		Lt Foot Dorsum	0900pm	Single	N
63	1.02	0500pm	Dhayalan	40	M	7127	Kanchipuram	Outdoor	Barefoot walking	Coolie		Lt Foot Dorsum	0600pm	Single	Y
64	12.04	0115am	Mary Margret	32	F	23571	Chennai	Outdoor	Sitting in front of house	House wife		**Rt Gluteal Region**	0700pm	Multiple	N
65	5.06	1230am	Govindammal	30	F	37024	Sriperampathur	Outdoor	Barefoot walking	Coolie		Rt Calf	0800pm	Multiple	N
66	4.08	1255pm	Munikrishnan	31	M	36576	Gumpidipoondi	Outdoor	Arranging things	Coolie	rt hand finger		0800am	Single	N
67	1.09	0900pm	Munusamy	60	M	59620	Arokonam	Outdoor	Bitter melon field	Agriculture	lt middle finger		1130am	Single	Y
68	30.08	0945pm	Kumar	22	M	59177	Kolathur	Outdoor	Barefoot walking	Student		Lt Middle Toe	0630pm	Single	Y
69	28.08	1230pm	Elizabeth Rani	32	F	58581	Sriperampathur	Outdoor	Sitting in temple	Export worker	lt middle finger		0900pm	Single	Y
70	7.05	0900pm	Venkatesan	28	M	29828	Near Chetinad Hospital	Outdoor	Barefoot walking	Coolie		Rt Big Toe	0800pm	Single	Y
71	27.05	0900pm	Balamurugan	25	M	34872	Thiruvallur	Outdoor	Open air toilet	Coolie		Lt Foot Dorsum	0600pm	Single	N
72	7.05	1150pm	Atchaya	24	M	29850	Gumpidipoondi	Outdoor	Open air toilet	Coolie		Lt Calf Reion	0630pm	Single	Y
73	6.05	1030pm	Ameresan	17	M	29526	Kanchipuram	Outdoor	Climbing tree	Agriculture	lt middle finger		0330pm	Single	Y
74	30.04	0300pm	Stanley Andrews	48	M	28101	Ambattur	Outdoor	Handling snake	Profe.snake.catch	rt arm		0115pm	Single	Y
75	28.05	1030pm	Meenakshi	25	F	35221	Tiruvanamalai	Outdoor	Barefoot walking	House wife		Rt Dorsum Foot	0430pm	Single	Y
76	19.04	545am	Babu	35	M	25303	Gumpidipoondi	Outdoor	Working in fields	Agriculture		Lt Dorsum Foot	0700pm	Single	Y
77	28.05	0600am	Manikandan	13	M	34911	Sriperampathur	Indoor	Sleeping	Student		Lt Dorsum Foot	0300am	Single	Y
78	27.04	0200pm	Rajendran	39	M	27471	Kanchipuram	Outdoor	Outside house	Agriculture		Rt Dorsum Foot	0730pm	Single	Y

Sr No	Dt of Admission	Time	Name	Age	Sex	IP No	Place	In/Outdoor	Work	Occupation	Site of Bite UL	Site of Bite LL	Time	No of Bite	ligature
79	15.03	0800pm	Renuka	32	F	16950	Chennai	Outdoor	Barefoot walking	Coolie		Lt Dorsum Foot	0600pm	Single	Y
80	15.03	0800pm	Janaki	55	F	17211	Chennai	Outdoor	Barefoot walking	Coolie		Rt Dorsum Foot	0600pm	Single	Y
81	22.03	0900pam	Sathishkumar	27	M	18654	Kanchipuram	Outdoor	Paddy field	Agriculture		Lt Dorsum Foot	0700pm	Single	Y
82	1.03	0700am	Vibhu	22	M	13894	Chennai	Outdoor	Barefoot walking	Student		Lt Dorsum Foot	0600am	Single	N
83	3.03	0900pm	Mohan	41	M	14193	Sriperampathur	Indoor	Sleeping	Agriculture	lt hand		0600pm	Single	Y
84	16.01	0445pm	Kullamma	75	F	3214	Villupuram	Outdoor	Working in fields	Agriculture		Lt Dorsum Foot	0600am	Single	Y
85	28.01	1140pm	Ayyamarappan	40	M	6064	Avadi	Outdoor	Barefoot walking	Government emplo		Rt Dorsum Foot	0700pm	Single	Y
86	23.02	0910pm	Vishvanathan	25	M	5012	Chennai	Outdoor	Barefoot walking	Student		Lt Dorsum Foot	0600pm	Single	N
87	13.02	1145am	Eshwaran	32	M	10071	Chennai	Outdoor	Barefoot walking	Government emplo		Lt Dorsum Foot	0945am	Single	N
88	15.02	0640pm	Gowi	18	F	10638	Ponneri	Outdoor	Sugarcane farm	Agriculture		Rt Dorsum Foot	0645am	Single	Y
89	17.02	1115am	Murugama	50	F	11001	Thiruvallur	Outdoor	Barefoot walking	Coolie		Lt Dorsum Foot	0715am	Single	N
90	23.02	0800pm	Asish	23	M	12563	Thiruvallur	Outdoor	Barefoot walking	Student		Rt Dorsum Foot	0800am	Single	N
91	27.02	0930pm	Pradeep	37	M	13365	Chennai	Outdoor	Around home	Government emplo		Lt Dorsum Foot	0630pm	Single	N
92	26.03	1000am	Shanmugam	40	M	19842	Thirutanni	Outdoor	Barefoot walking	Coolie		Rt Dorsum Foot	0600am	Single	N
93	18.03	0900pm	Bagiyalakshmi	68	F	25273	Thiruvanamalai	Outdoor	Working in fields	Agriculture	lt hand		0900am	Single	Y
94	22.04	0900pm	Thyagu	16	M	26145	Thiruvallur	Outdoor	Barefoot walking	Student		Lt Dorsum Foot	0600pm	Single	N
95	27.04	0800am	Rajendran	38	M	27471	Kanchipuram	Outdoor	Paddy field	Agriculture		Lt Dorsum Foot	0800am	Single	Y
96	9.05	1100am	Yabish	19	M	30395	Thiruvallur	Outdoor	Barefoot walking	Student		Rt Dorsum Foot	0900am	Single	N
97	17.05	0900pm	Sivakumar	25	M	32226	Chennai	Outdoor	Barefoot walking	Government emplo		Rt Dorsum Foot	0600pm	Single	N
98	21.05	0900pm	Rajeshwari	39	F	33080	Chennai	Outdoor	Barefoot walking	House wife		Rt Dorsum Foot	0700pm	Single	N
99	3.06	1000am	Poongan	38	M	36594	Thiruvanamalai	Outdoor	Sugarcane farm	Agriculture		Lt Dorsum Foot	0800am	Single	Y

Sr No	Dt of Admission	Time	Name	Age	Sex	IP No	Place	In/Outdoor	Work	Occupation	Site of Bite UL	Site of Bite LL	Time	No of Bite	ligature
100	9.06	0600pm	Munusamy	75	M	38201	Thiruvallur	Outdoor	Paddy field	Agriculture		Rt Dorsum Foot	0600pm	Single	Y

First Aid Method	Firstaid physcian/nonphyscian	Private(pr)/ government(g)/native(na)	First symptom	Time of Apearance	Type of Snake	Time of First dose of ASV	Bite to GGH Admission	Reason for delay	Neurotoxicity / hemotoxicity/ Conmbined	Regional Hymphadenopathy	Llocal Envenomation	Ptosis	Respiratory paralysis
	P	G	Vomiting	1h	Russel	1h	8h	Kept Under Observation	C		Y		
	P	PR	Ptosis	6h	Krait	6h	2h30m		N		N	6h	Breathing difficulty
		G			Non poisonous		4h						
	P	G			Non poisonous		6h						
	P	G			Nonpoisonous		4h				N-localised swelling		
	P	G	Vomiting	3h	Russels	4h	144h	Treated At Madurai	H	Y	Y		
	NP	NA	Vomiting	1h	Russels	98h	98h	Treated Native Medicine	H		Y		
Leaves	NP	NA	Gum Bleed	4h	Russels	12 h	98h	Treated In Andra	C		Y	4h	
	P	G	Blurring Of Vision	1h	Russels	2h	48h	Treated In Tiruvanamalai	H	Y	Y		
	P	PR	Blurring Of Vision	1h	Russels	2h	36h	Treated In Chengalpattugh	H		Y		
Powder	NP	NA	Blurring Of Vision	1h	Russels	2h	36h	Treated In Vandavasi	C	Y	Y	3h	
Powder	NP	NA	Hematuria	17h	Russels	27h	27h	Observed In Home	Non poisonous		N		
	P	G	Prolonged Clotting	1 h	Russels	1h	14h	Treated In Arokonam Gh	H		Y		
Leaves	NP	NA	Prolonged Clotting	1hr	Russels	1h	56h	Treated In Tiruvanamalai	C	Y	Y	24h	
Leave Uice	NP	NA	Bleeding Gums	30min	Russels	1h	4h30m	Treated In Tiruvallur Gh	H		N		
Leaves	NP	NA	Dizziness	25min	Saw scaled	1h 25 min	12h	Shutteled Through Various Hospitals	H		Y		
Leaves	NP	NA	Bleeding Gums	6h	Saw scaled	10hr	10hr	Kept In Ponerigh	H	Y	Y		
Leaves	NP	NA	Blurring Of Vision	1h	Russels	1h30min	7h30min	Thiruvallur Gh	C	Y	Y	1h30min	
	P	G	Ptosis	2h	Russels	2h	7h30min	Tiruvallur Gh	C		Y	2h	
Powder	NP	NA	Gum Bleed	1h	Russels	3h	3h	Travelling	C	Y	Y	1h30min	

First Aid Method	Firstaid physcian/nonphyscian	Private(pr)/ government(g)/native(na)	First symptom	Time of Apearance	Type of Snake	Time of First dose of ASV	Bite to GGH Admission	Reason for delay	Neurotoxicity / hemotoxicity/ Conmbined	Regional Hymphadenopathy	Llocal Envenomation	Ptosis	Respiratory paralysis
	P	PR	Ptosis	21h	Russels	24h	24h	Went Home After Getting First Aid	C	N	Y	21h	
	P	G	Clooting Time Incre	9h	Sawscaled	9h	19h	Under Observation At Arakonam Gh	H	N	Y		
Leaves	NP	NA	Headache	4h	Sawscaled	12h	12h	Travelling	H	Y	Y		
	NP	NA	Bleeding Gums	2h	Russels	10h	5days	Treated In Tiruvanamalai	C	Y	Y	y	
	P	PR	Bleb	2h	Russels	8h	3h	Travelling	H	Y	Y		
	P	G	Ptosis	2h	Russels	4h	4h	Travelling	C	Y	Y	2h	
Leaves	NP	NA	Persistent Pain	immediate	Russels	4h	12h	Treated In Kancheepuram Gh	H	Y	Y		
Leaves	NP	NA	Vomiting	1h	Russels	2h	12h	Travelling	C	Y	Y	y	
	P	G	Blurring Of Vision	4h	Sawscaled	4h	12h	Treated In Ponneri Gh	H	N	Y		
	P	G	Hemetemesis	2h	Russels	2h	9h	Treated In Tiruvanamalai	H	N	Y		
	P	PR	Bleeding Gums	4h	Sawscaled	6h	6h	Was In Home	H	Y	Y		
Leaves/Powder	NP	NA	Double Vision	3h	Cobra	11h	11h	Bus Facility Not Available	N	Y	Y		
	P	G	Prolonged Clotting	2h30m	Sawscaled	2h30m	24h	Treated In Arokonam Gh	H	N	Y		
	P	G	Prolonged Clotting	1h	Sawscaled	1h	1h	Direct	H	Y	Y		
	P	G	Ptosis	12h	Krait	12h	33h	Treated In Jipmer	N	N	N	24h	
	P	G	Ptosis	1h	Russels	1h	5h	Travelling	C	Y	Y	1h30min	
	P	G	Ptosis	30min	Cobra	1h	5h	Travelling	N	N	Y	30m	
Leaves	NP	G	Dysphonia	30min	Cobra	3h	3h	Travelling	H	N	Y	1h	Y
Leaves	NP	NA	Ptosis	1h	Krait	8h	8h	Tavelling	H	N	N	1h	

First Aid Method	Firstaid physcian/nonphyscian	Private(pr)/ government(g)/native(na)	First symptom	Time of Apearance	Type of Snake	Time of First dose of ASV	Bite to GGH Admission	Reason for delay	Neurotoxicity / hemotoxicity/ Conmbined	Regional Hymphadenopathy	Llocal Envenomation	Ptosis	Respiratory paralysis
	P	G	Ptosis	1h	Krait	1h	3h	Travelling	N	N	N	1h	
Powder	NP	NA	Double Vision	1h30m	Cobra	1h30min	10hr	Treated At Ponneri	N	Y	Y		
Powder/Leaves	NP	NA	Ptosis	6h	Cobra	8h	8h	Native Treatement	N	N	Y	6h	
Leaves	NP	NA	Ptosis	3h30m	Russels	7h	7h	Native Treatement	C	Y	Y	3h30m	
	P	PR	Ptosis	30min	Cobra	2h	6h	Treated At Private	N	N	Y	30m	
Leaves	NP	NA	Ptosis	9h	Cobra	20h	20h	Native Treatement	N	Y	Y	9h	
Liquid	NP	NA	Blurring Of Vision	1h	Krait	2h	5h	Treated At Ponneri	N	N	Y		
	P	G	Double Vision	1h30m	Cobra	1h30min	45m	Direct	N	N	Y	2h	
Leaves	NP	NA	Ptosis	1h	Cobra	15h	21h		N	N	Y	1h	
Leaves	NP	NA	Ptosis	1h	Cobra	2h30m	2h30m		N	Y	Y	1h	
Leaves	NP	NA	Ptosis	45m	Cobra	1h	5h		N	Y	Y	45m	
Snake Stone	NP	NA	Ptosis	12h	Russels	18h	36h		C	N	Y	12h	
	P	G	Prolonged Clotting	2h	Russels	2h	43h		H	Y	Y		
	P	G	Local Envenomation	1h	Saw scaled	5h	24h		Local	N	Y		
	P	PR	Prolonged Clotting	2h	Saw scaled	6h	6h		H	Y	Y		
	P	G	No Symptoms		Nonpoisonous		3h		Non poisonous	N	N		
	P	G	No Symptoms		Nonpoisonous		2h		Non poisonous	N	N		
	P	G	No Symptoms		Nonpoisonous		1h30m		Non poisonous	N	N		
	P	G	No Symptoms		Nonpoisonous		3h		Non poisonous	N	N		

First Aid Method	Firstaid physcian/nonphyscian	Private(pr)/ government(g)/native(na)	First symptom	Time of Apearance	Type of Snake	Time of First dose of ASV	Bite to GGH Admission	Reason for delay	Neurotoxicity / hemotoxicity/ Conmbined	Regional Hymphadenopathy	Llocal Envenomation	Ptosis	Respiratory paralysis
	P	PR	No Symptoms		Nonpoisonous		5h		Non poisonous	N	N		
	P	PR	No Symptoms		Nonpoisonous		2h		Non poisonous	N	N		
Leaves	NP	NA	Ptosis	2h	Krait	2h	2h		N	N	N	2h	
	P	PR	No Symptoms		Nonpoisonous		12h		Non poisonous	N	N		
	P	PR	Prolonged Clotting	2h	Sawscaled	2h	47h		H	N	Y		
	NP	NA	No Symptoms		Nonpoisonous		6h		Non poisonous	N	N		
	P	G	No Symptoms		Nonpoisonous		3h30m		Non poisonous	N	N		
	P	PR	No Symptoms		Nonpoisonous		5h		Non poisonous	N	N		
Powder	NP	NA	Prolonged Clotting	1h30m	Russels	1h30min	9h	Treated At Arokoam	H	N	Y		
	P	PR	Prolonged Clotting	5h	Russels	5h	3h	Travelling	H	N	N		
	P	PR	Ptosis	4h	Krait	15h	15h	Observed At Private Hospital	N	N	N	4h	
Leaves	NP	NA	Hemetemesis	8h	Sawscaled	25h	25h	Went Home After Getting First Aid	H	N	Y		
	P	PR	No Symptoms		Nonpoisonous		3h	Travelling	Non poisonous	N	N		
	P	G	Dysphonia	2h	Cobra	5h	5h	Travelling	N	Y	N		
Leaves/Powder	NP	NA	Dysphonia	30min	Cobra	2h	7h	Travelling	N	Y	Y	8h	
	P	G	Ptosis	7h	Cobra	no asv	2h	Travelling	Dry bite	N	Y	7h	
	P	PR	Giddiness	6h	Nonpoisonous	no asv	6h	Travelling	Non poisonous	N	N		
	P	G	Ptosis	1h	Russels	10h	10hr	Treated In Private	C	N	Y	1h	
	P	G	Dysphonia	2h	Cobra	3h	3h	Travelling	N	Y	Y	3h	
Leaves	NP	NA	Local Envenomation	1h	Cobra	1h	5h	Travelling	Local	N	Y		

First Aid Method	Firstaid physcian/nonphyscian	Private(pr)/ government(g)/native(na)	First symptom	Time of Apearance	Type of Snake	Time of First dose of ASV	Bite to GGH Admission	Reason for delay	Neurotoxicity / hemotoxicity/ Conmbined	Regional Hymphadenopathy	Llocal Envenomation	Ptosis	Respiratory paralysis
	P	PR	Ptosis	1h	Cobra	2h	2h	Travelling	N	Y	Y	1h	
	P	PR	No Symptoms		Nonpoisonous		2h	Travelling	Non poisonous	N	N		
Leaves	NP	NA	Bleeding Gums	3h	Russels	6h	72h	Treated At Chengalpattu	C	Y	Y	5h	
	P	PR	No Symptoms		Nonpoisonous		1h	Travelling	Non poisonous	N	N		
Leaves	NP	NA	Ptosis	3h	Krait	3h	27h	Treated In Kancheepuram Gh	N	N	N	3h	
Leaves	NP	NA	Ptosis	4h	Russels	4h	10hr	Treated In Villupuram	C	Y	Y	4h	
	P	NA	Ptosis	2h	Cobra	4h	4h	Travelling	N	N	Y	2h	
	P	PR	No Symptoms		Nonpoisonous		3h	Travelling	Non poisonous	N	N		
	P	PR	No Symptoms		Nonpoisonous		j2h	Travelling	Non poisonous	N	N		
Leaves	NP	NA	Ptosis	1h	Cobra	3h	12h	Treated In Ponneri Gh	N	N	Y	1h	
Leaves	NP	NA	No Symptoms		Nonpoisonous		4h	Travelling	Non poisonous	N	N		
	P	PR	Ptosis	2h	Krait	3h	12h	Treated In Thiruvallur	N	N	N	2h	
	P	PR	No Symptoms		Nonpoisonous		3h	Travelling	Non poisonous	N	N		
	P	PR	No Symptoms		Nonpoisonous		4h	Travelling	Non poisonous	N	N		
Leaves	NP	NA	Bleeding Gums	3h	Russels	5h	36h	Treated In Tiruvanamalai	C	Y	Y	6h	
	P	PR	No Symptoms		Nonpoisonous		3h	Travelling	Non poisonous	N	N		
Powder	NP	NA	Bleeding Gums	4h	Russels	6h	30h	Treated In Kancheepuram Gh	H	Y	Y		
	P	PR	Ptosis	2h	Krait	5h	5h	Travelling	N	N	N	2h	
	P	PR	No Symptoms		Nonpoisonous		3h	Travelling	Non poisonous	N	N		
	P	PR	No Symptoms		Nonpoisonous		2h	Travelling	Non poisonous	N	N		
Leaves	NP	NA	Bleeding Gums	2h	Russels	4h	26h	Treated In Tiruvanamalai	H	Y	Y		

First Aid Method	Firstaid physcian/nonphyscian	Private(pr)/ government(g)/native(na)	First symptom	Time of Apearance	Type of Snake	Time of First dose of ASV	Bite to GGH Admission	Reason for delay	Neurotoxicity / hemotoxicity/ Conmbined	Regional Hymphadenopathy	Llocal Envenomation	Ptosis	Respiratory paralysis
Powder	NP	NA	Bleeding Gums	1h	Russels	12h	48h	Went Home After Getting First Aid	C	Y	Y	4h	

Bleeding gums	Hematuria	Hemetemesis	Neostigmine res.	Other syptoms	B.urea(mgm/dl)	Sr.Creatinine (mgm/dl)	Potassium (meq/dl)	Hemoglobin	SAP	Ventilatory support	Dialysis	Blood products	No of ASV used	ARF	Outcome	Period of stay	Point of interest/specific symtom
				Dysphonia/dysphagia	18	1	3	11	94	N	N	N	18		Discharged	5	Persistent edema
			no		28	0.9	3.4	11.5	76	N	N	N	18		Discharged	5	Intense myalgia,LDH 735
				Giddiness only	34	0.7	4	10	78	N	N	N	NO ASV		Discharged	2	Giddiness
				No symptoms	21	0.7	3.1	13	97	N	N	N	NO ASV		Discharged	2	Dry bite by poisonous snake
				No symptoms	18	0.9	3.8	12.2	345	N	N	N	NO ASV		Discharged	3	
				Presented with arf-olig	165	12	3.3	10	126	N	Peritoneal	N	18	Yes	Discharged	14	
				Presented with arf-olig	187	15	5	9	176	N	Peritoneal	N	18	Yes	Death	12	Late presentation
4h	12h			Presented with arf-olig	223	18.7	3.9	11.5	75		Hemodialysis	N	18	Yes	Discharged	13	Late presentation
				Presented with arf-olig	150	2.8	4.5	10	159	N	Hemodialysis	Ffp/platelets	18	Yes	Discharged	14	Ref original
	12h			Presented with arf-olig	79	2.8	4.4	12	155	N	Hemodialysis	N	18	Yes	Discharged	11	
				Presented with arf-olig	90	5.3	4.8	4.4	83	Y	Hemodialysis	Ffp/platelets	18	Yes	Death	16	Arf/dic/schock
					122	5.7	5.4	10	65	N	Hemodialysis	N	NO ASV	Yes	Discharged	13	Myocar. Infarct inf wall with arf.no asv
					32	1	4	13	76	N	N	N	8		Discharged	3	Low dose
				Presented with arf-olig	143	5	4.6	12.3	148	N	Hemodialysis	N	18	Yes	Discharged	11	
					32	1	4.2	11	98	N	N	N	18		Discharged	2	Unconsious immediately -pain
				Only clotting time prolonged	32	1.2	3.2	10	89	N	N	Ffp	18		Discharged	4	Pregnant 9mon
6h					34	2.2	3.4	12	100	N	N	N	18	N	Discharged	3	
					22	1.5	3.6	9.5	154	N	N	N	18	N	Discharged	2	
			no	Clotting time pro.1h30m	31	1	2.1	13	140	N	N	N	18	N	Discharged	1	
	6h		no	All manifestation	37	1.1	3.1	13	234	N	N	Ffp/platelets	18	N	Discharged	4	Total billurubin

Bleeding gums	Hematuria	Hemetemesis	Neostigmine res.	Other syptoms	B.urea(mgm/dl)	Sr.Creatinine (mgm/dl)	Potassium (meq/dl)	Hemoglobin	SAP	Ventilatory support	Dialysis	Blood products	No of ASV used	ARF	Outcome	Period of stay	Point of interest/specific symtom
				Altered taste	19	0.8	3.6	9.6	140	N	N	N	18	N	Discharged	1	
					30	0.8	3.4	12.5	140	N	N	N	18	N	Discharged	1	
				Clotting time prolonged	20	0.9	3.7	12	154		N	N	5	N	Discharged	2	
	y		no	Presented with arf-olig	176	5.5	5.3	10	243	N	Hemo+perito	Ffp/platelets	18	Yes	Ama	10	Dic/renal failure/toxic optic neuritis
				Bleb in 2hr	24	1.2	3.6	14.4	160	N	N	N	13	N	Discharged	2	Discolouration
			no	Eom restricted	29	0.9	2.9	15.5	154	N	N	N	13	N	Discharged	1	
					22	1	3.6	12.3	111	N	N	N	18	N	Discharged	2	
	y		no	Heamaturia only	34	1.1	3.8	12	165	N	N	N	18	N	Discharged	2	
				Blurring +ct prolonged	40	1.2	3.8	11.8	156	N	N	N	18	N	Discharged	1	
				Gumbleed,hematuria,othrs	144	4	4.5	10	176	N	N	Ffp/platelets	18	Yes	Death	1	Loss of vision(optic neuritis,viterous hemorrhage),sluggish
				Lymphadenitis first 1h	33	1	3.6	10.8	21	N	N	Ffp	18	N	Discharged	2	Lymphadinitis first,profuse gum
				Tansient double vision	18	0.7	4.3	9.3	116	N	N	N	8	N	Discharged	3	
				No symptoms	39	1.2	3.8	13	379	N	N	N	6	N	Discharged	1	
				Exremely pain lymphade	28	0.7	3.2	14	85	N	N	N	13	N	Discharged	3	Extremely painful lymphadenopathy
				Cent.pon. Myelinolysis	100	1.9	3.2	11	211	Y(3days)	N	Ffp/platelets	18	N	Death	22	Alcoholic,prolonged ventilation,hepatitis like picture(AST/ALT-
			no	Prolonged clotting	22	0.8	3.1	14	215	Y(24h)	N	N	18	N	Discharged	6	Ldh-1465
			yes	Dysphonia/dysphagia	34	1	2.7	11.8	187	N	N	N	18	N	Discharged	3	
			yes	Prolonged bite	25	0.8	3.5	12.5	92	Y(36h)	N	N	18	N	Discharged	2	
				Dysphonia/opthalmoplegia	34	1	3.9	13	98	Y(12h)	N	N	18	N	Discharged	2	

Bleeding gums	Hematuria	Hemetemesis	Neostigmine res.	Other syptoms	B.urea(mgm/dl)	Sr.Creatinine (mgm/dl)	Potassium (meq/dl)	Hemoglobin	SAP	Ventilatory support	Dialysis	Blood products	No of ASV used	ARF	Outcome	Period of stay	Point of interest/specific symtom
				Giddiness and headache	35	1	4	10	95	N	N	N	12	N	Discharged	1	
				Excruciating pain	23	1.1	3.7	12	87	N	N	N	18	N	Discharged	2	
				Persistant vomiting	32	0.9	4.1	14.7	256	N	N	N	18	N	Discharged	2	
				Prolonged clotting time	134	1.2	3.9	12.8	176	N	N	Ffp/platelets	18	N	Discharged	7	Transient rise of RFT,subsided without dialysis
			yes	Gluteal welling	32	1	3.5	11	306	N	N	N	18	N	Discharged	2	
			yes		33	0.8	4.1	12.4	179	N	N	N	18	N	Discharged	5	
			no	No asv at ggh	28	1.4	3.6	11.3	184	N	N	N	14	N	Discharged	2	No asv at ggh
			yes	Direct admission	25	0.7	3.6	15.7	156	Y(24h)	N	N	13	N	Discharged	4	Improved over 24 h
			yes		70	2.4	3.9	14	175	Y(24h)	N	N	18	N	Discharged	11	Transient rise of RFT,subsided without dialysis
			yes		22	0.7	0.4	9.8	101	Y(4h)	N	N	18	N	Discharged	5	Cpk 706,cpkmb 58,ldh 583
			yes	Breathing dificulty	22	1	3.8	11.4	176	N	N	N	18	N	Discharged	5	Blurring of vision for 6 days
			no		98	2	4.2	10.7	198	N	N	N	18	Yes	Discharged	3	Diplopia and numbness persisting
				Lotting only prolonged	26	1.2	3.3	13.7	200	N	N	N	18	N	Discharged	2	Immediate unconsiousness(pain shock),clotting only prolonged
					24	1	3.6	8.5	152	N	N	N	10	N	Discharged	2	Only local envenomation
				Clotting prolonged	31	1	3.4	12	146	N	N	N	8	N	Discharged	1	Transient blurring of vision
					22	0.9	3.8	11	78	N	N	N	NO ASV	N	Discharged	1	
					25	0.9	4	11.6	87	N	N	N	NO ASV	N	Discharged	1	
				No symptoms	21	0.7	3.4	10.6	31	N	N	N	NO ASV	N	Discharged	2	
				No symptoms	22	0.7	4	13	74	N	N	N	NO ASV	N	Discharged	1	

Bleeding gums	Hematuria	Hemetemesis	Neostigmine res.	Other syptoms	B.urea(mgm/dl)	Sr.Creatinine (mgm/dl)	Potassium (meq/dl)	Hemoglobin	SAP	Ventilatory support	Dialysis	Blood products	No of ASV used	ARF	Outcome	Period of stay	Point of interest/specific symtom
				No symptoms	17	0.8	3	15	76	N	N	N	NO ASV	N	Discharged	1	
				Headache	26	0.8	3.1	12	105	N	N	N	NO ASV	N	Discharged	1	
			no	Headache	23	0.7	3.6	10.6	196	N	N	N	8	N	Discharged	1	
				No symptoms	17	0.7	4	13.8	144	N	N	N	NO ASV	N	Discharged	2	
				Hemo+edema	32	1	3.7	13	97	N	N	N	10	N	Discharged	2	
				Local edema	17	0.7	3	15	63	N	N	N	NO ASV	N	Discharged	1	
				Local edema	20	0.9	3.2	11.5	178	N	N	N	NO ASV	N	Discharged	1	
				Local edema	19	1	3.8	10.9	112	N	N	N	NO ASV	N	Discharged	2	
					29	0.7	3.6	13.9	85	N	N	N	10	N	Discharged	2	
				No local edema,lynadeno	19	0.8	3.9	10.6	194	N	N	N	8	N	Discharged	2	
				No local edema,lynadeno	18	0.6	3.3	10.6	98	N	N	N	18	N	Discharged	3	
				Epistasis,	32	0.7	3.7	12	154	N	N	N	14	N	Discharged	2	
					17	0.7	3.2	8	236	N	N	N	NO ASV	N	Discharged	2	
				No ptosis	21	0.8	3.8	12.3	118	Y(24h)	N	N	18	N	Discharged	4	
				Late ptosis	17	0.8	3.5	11.1	187	N	N	N	13	N	Discharged	2	
				Transient ptosis	19	0.9	4.3	13	112	N	N	N	NO ASV	N	Discharged	2	Transient ptosis,no asv
					26	1	3.6	10	93	N	N	N	NO ASV	N	Discharged	1	
				Arf	87	3.3	5.4	10	187	N	Hemo		18	Yes	Discharged	9	
				Compartmental syndrome	21	0.9	3.2	11	431	N	N	N	18	N	Discharged	2	Compartmental syndrome
					50	1.4	4.1	13.5	82	N	N	N	14	N	Discharged	2	

Bleeding gums	Hematuria	Hemetemesis	Neostigmine res.	Other syptoms	B.urea(mgm/dl)	Sr.Creatinine (mgm/dl)	Potassium (meq/dl)	Hemoglobin	SAP	Ventilatory support	Dialysis	Blood products	No of ASV used	ARF	Outcome	Period of stay	Point of interest/specific symtom
				Compartmental syndrome	32	1.1	3.7	12.1	108	N	N	N	18	N	Discharged	3	
				Local edema	22	0.7	3.8	11.1	67	N	N	N	NO ASV	N	Discharged	1	
3h				Presented with arf-olig	98	4.5	5	10	199	N	Hemo	N	18	Yes	Discharged	18	
				Local edema	20	0.8	3.8	13	76	N	N	N	NO ASV	N	Discharged	2	
				No local edema,lynadeno	33	1.1	4.1	12	136	N	N	N	18	N	Discharged	1	
				Clotting prolonged	43	1.2	3.6	11	218	N	N	N	18	N	Discharged	4	
					21	1.1	4.1	10.7	165	N	N	N	13	N	Discharged	2	
				Local edema	20	1	3.5	10.9	87	N	N	N	NO ASV	N	Discharged	2	
				Local edema	20	1.3	3.6	12	76	N	N	N	NO ASV	N	Discharged	1	
			yes		34	1.1	4.1	10	66	N	N	N	13	N	Discharged	1	
				Local edema	31	1	3.9	11	84	N	N	N	NO ASV		Discharged	1	
				No local edema,lynadeno	28	1.1	4.2	13	191	N	N	N	18	N	Discharged	4	
				Local edema	22	1	4.1	14	65	N	N	N	NO ASV	N	Discharged	1	
				Local edema	30	1.1	3.9	12	56	N	N	N	NO ASV	N	Discharged	1	
					41	1	4.1	10	231	N	N	N	18	N	Discharged	1	
				Local edema	23	0.8	3.9	14	46	N	N	N	NO ASV	N	Discharged	1	
					41	1.4	4	11	201	N	N	N	18	N	Discharged	2	
				No local edema,lynadeno	22	1	3.8	12	76	N	N	N	NO ASV	N	Discharged	3	
				Local edema	31	1.2	4.1	14	48	N	N	N	NO ASV	N	Discharged	1	
				Local edema	30	1	3.9	12	65	N	N	N	NO ASV	N	Discharged	1	
					43	1.3	4	14	169	N	N	N	18	N	Discharged	2	

Bleeding gums	Hematuria	Hemetemesis	Neostigmine res.	Other syptoms	B.urea(mgm/dl)	Sr.Creatinine (mgm/dl)	Potassium (meq/dl)	Hemoglobin	SAP	Ventilatory support	Dialysis	Blood products	No of ASV used	ARF	Outcome	Period of stay	Point of interest/specific symtom
				Presented with arf-olig	104	4	5.4	10	261	N	Hemo	Ffp/platelets	18	Yes	Death	8	