"STUDY ON CLINICAL PROFILE OF SNAKE BITE WITH SPECIAL REFERENCES TO NEUROLOGICAL AND HEMATOLOGICAL COMPLICATIONS"

By

Dr MOHD TANVEER ULLA KHAN

SDUAHER

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IN

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Under the guidance of

Dr V.Lakshmaiah, M.D(Gen Med).DCH
HOD & Professor

DEPARTMENT OF GENERAL MEDICINE
SRI DEVARAJ URS MEDICAL COLLEGE
KOLAR - 563101

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is a bonafide and genuine research work carried out by me under the guidance of **Dr.V.Lakshmaiah., M.D. H.O.D & Professor,** Department Of General medicine, Sri Devaraj Urs Medical College, Tamaka, Kolar.

Dr.Mohd Tanveer

Date:

Place : Kolar .

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE, TAMAKA , KOLAR , KARNATAKA,

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Dr.V.Lakshmaiah, M.D(Gen med),dch.

H.O.D & Professor

Department Of General medicine

Sri Devaraj Urs Medical College,

Tamaka,Kolar.

Date :

Place : Kolar

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE, TAMAKA , KOLAR , KARNATAKA,

ENDORSEMENT BY THE HOD,

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Dr. V.LAKSHMAIAH.
Professor and HOD,
Department Of General medicine
Sri Devaraj Urs Medical College,
Tamaka,Kolar.

Dr.SANIKOP M.B,
Principal,
Sri Devaraj Urs Medical college
Tamaka,Kolar.

Date:
Place: Kolar

Date:
Place: Kolar

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE, TAMAKA , KOLAR , KARNATAKA,

ETHICAL COMMITTEE

CERTIFICATE

This is to certify that the Ethical committee of Sri Devaraj Urs Medical College, Tamaka, Kolar has unanimously approved

Dr. **Mohd Tanveer** Post-Graduate student in the subject of GENERAL MEDICINE at Sri Devaraj Urs Medical College, Kolar to take up the Dissertation work entitled

"STUDY ON CLINICAL PROFILE OF SNAKE BITE WITH SPECIAL REFERENCES TO NEUROLOGICAL AND HEMATOLOGICAL COMPLICATIONS"

to be submitted to the SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH CENTRE, TAMAKA , KOLAR , KARNATAKA,

Member Secretary
Sri Devaraj Urs Medical College,
Kolar–563101

Date :

Place : Kolar

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Dr. Mohd Tanveer

Date :

Place : Kolar

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LIST OF ABBREVATIONS USED :

A - Absent

ASV - Anti Snake Venom

DMA - Discharge against Medical Advice

DC - Differential Court

ECG - Electrocardiogram

F - Female

M - Male

P - Present

SB - Sinus Bradycardia

ST - Sinus Tachycardia

TC - Total Count

WNL - Within Normal Limit

BT- Bleeding time

Hb-Hemoglobin

ABSTRACT:

AIMS AND OBJECTIVES:

Snake bite is a major public health problem throughout the world especially in tropical and subtropical countries.

The objective of the study is to study the clinical profile of snake bite and to evaluate the neurological and hematological complications of snake bite.

METHODS AND MATERIALS:

The cases for present study were taken from the Department of General Medicine, R.L. Jalappa .Hospital,. The cases were studied from DEC 01 2008 onwards till NOV 30 2009 of snake bite and its hematological and Neurological Complications were completed. 79 snakebites were carefully examined and monitored for hematological and Neurological complications, and were encounted.

It was a Hospital case series study in which 50 patients who gave history of snake bite or had signs of envenomation or fang marks were selected.

RESULTS AND ANALYSIS

The total number of snake bite admissions in the medical wards during that period was 79 out of which 50 cases were taken up for the study out of which 26% had hematological and neurological complications. Local envenomation was seen in 34%. No envenomation in 14%.

There were 70% of male victims compared to 30% females. 70% of the bite were between 12:00pm-11:59pm. 62% of cases were seen in rainy season. Most of the

cases were from rural population during working in the fields. Most of the people came to hospital with in 8 hrs of bite.The most common site of bite was foot accounting for more than 80%.Ptosis and diplopia were the commonest neurological manifestation and hematuria and bleeding gums the common hematological manifestation.

CONCLUSION :

Snake bites are common in and around Kolar , Majority of them presents with neurotoxic and hematotoxic manifestations, Among species Cobra and viper are the commonest ptosis,diplopia, bleeding gums,were the commonest and earliest manifestation. If ASV is administered at the earliest complications can be avoided.

TABLE OF CONTENTS

INTRODUCTION

Snakebite continues to be equally as mysterious as the snakes, since the dawn of human civilization. In any part of the world, snakes produce an un imaginable fear and anxiety. Snakebite may be earliest and the most common poisoning known to human benig. Myths in forms of stories, folk tales, dramas and films revolve around the dreaded snakes, rivalry of snakes, mating of the snakes. Snakes are worshipped in India and auspicious days are marked in the name of snake's [1.2].

Snakes -are legless cold blooded reptiles. Of the 2500-3000 species of snakes, about 500 belong to the four families of venomous snakes and only about 200 species have caused death or permanent disability by biting humans. Approximately about 3 lakhs people are bitten by snake every year and about 15000 people in India. About 500 people are bitten by poisonous snakes in Karnataka[3.4].

Poisonous snakebite cause potential threat by causing neurotoxicity and ototoxicity (55). Various studies have shown hematological complications ranging 30% to 80% of the poisonous snakebites. In most of the Indian studies hematological complications are the most common complications of poisonous snakebites[4.23,50.] Various Hematological complications like bleeding from the site of bite, echymosis, hematuria, gum bleeding, melena, hemoptysis and intracranial hemorrhage are seen. Rare are subconjunctival, retro-orbital, sublingual, retinal and retroperitoneal bleeds.

Various investigations are done to diagnose hematological complications at the earliest with advent of immunological methods the diagnosis and the management of the snakebites have changed drastically in Western countries[14]. But in third world countries like ours with predominant rural set up it is not possible to carry out the investigations discussed by Western authors. Hence a practical approach has to be

adopted to diagnose, to treat and to prevent complications such as hematological and neurological manifestations in poisonous snakebites.

The present study is intended to study the hematological complications and neurological complication, diagnosis and management in poisonous snakebites. A hospital case series study was conducted in the department of Medicine, R. L. J. H, Tamaka , Kolar which is a teaching Hospital . The study included the collection of data of poisonous snakebites causing hematological complication and Neurological complication including age incidence, sex ratio, seasonal variations, first aid methods employed by the local people doctors and we have also included epidemiological back *up* for the study. Extensive clinical work up has been done in the patients having hematological complications.

Hematological investigations are chosen such that the investigations chosen are easily available, cheap, reproducible, reliable and informative. The investigations were intended to diagnose the hematological complications at the earliest and to monitor the treatment. Record of the prognosis was also made in all the cases to calculate mortality and morbidity.

AIMS AND OBJECTIVES

1. To study the clinical profile of snake bite
2. To evaluate the neurological and hematological complications associated with snake bite

REVIEW OF LITERATURE

HISTORICAL ASPECTS

Snakebite is known to man since immemorial. With advent of civilization various treatments have been tried for poisonous snakebites. Hematological and neurological complication of snakebite have been recorded in ancient civilization and so many myths have been attached to it.[23]

Various herbs and other methods have been tried for the management of snakebite in all countries. Management of snakebite victims has been attempted since the advent of medicine and it is found in various 'Materia media' including the earliest by Chinese Pan Tsao (BC 2700).

India snakes are worshiped and management was mainly based upon the myths attaached to it. First aid and treatment have been described in 'Rigveda and aveda' (BC 2500 - 3000) [10,11.] Sushrutha (5th AD) described first aid nes like Mahagad, Ajtagad and Rishabagad for the treatment of snake bite

All the previous modes of management were based solely upon the superstitions and myths. The logical and scientific approach for the management has started only in 18th century. Modern investigations and treatment of snakebites began by Abbabi Felice Fontana (1730 - 1803). Patrick Russell of id (1727 - 1805) wrote "An account of Indian serpents" and Sir Joseph (1824-1907) wrote Thanatophidia of India

Edward Jenner introduced the technique of vaccination and the first antivenin was described by Van Behring and Kitasato in 1890[28]. Since then venoms have been produced and have proved their efficacy. The complication of snakebite such as hematological complications are treated mainly by knowledge of hemostasis [29]

In 20th century attempts were made to explore the immunological variable for the development of antisera.[16J] Also there is invention of immunization against the snakebites. With still advances to come the management will take a new horizon in this century.

EPIDEMIOLOGY

Tropical countries where snakebite is a serious problem there are few reliable data. Records of the patients treated by traditional methods are lost to the official statistics**.** Hospital records, the sole source of most snakebite reporting are likely to over represent the more seriously envenomed patients, and depend on the enthusiasm and work load of the hospital.[23]

The victims of snake bites are mainly of the rural population, who are bitten during field work and when sleeping outdoors.[75]

The urbanization and deforestation, snakebite has become an important public health hazard. It is estimated that around 40000 - 50000 die of snakebite annually all over the world. India along with the South East Asian countries accounts for 85% of all reported snakebites in the world and 10-15 cases end in mortality.[40]

Though definite statistics of deaths due to snakebite is not maintained in India, it is believed that about 2 million indians are bitten by snakes every year and about 25000 to 60000 cases prove fatal. An estimated 15,000 -20,000 people die each year from snakebite in india [(59)] Approximately 8000 poisonous snakebites per year are reported in USA[(49)]. Poisoning from snakebite is an important medical emergency in Africa, South America, Indian subcontinent and South East Asia.

The annual death rate from snakebite in Brazil is 4/ 100000: in Burma 15.4/ 100000. In USA about 2000 die every year. In Europe death from snakebite is rare. In India an estimation of 100 deaths/day

Snakebite is an occupational hazard of the agricultural workers, herdsmen and hunter gatherer tribes and snake charmers in many tropical countries. Venomous snakes occur up to altitudes of 4000 meters in many parts of the world.[74] Poisonous snakebites are common and severe in early spring. Most of the snakebites are nonpoisonous. Out of

poisonous bites 80% are due to vipers, 10% are due to cobras, and kraits account for 4%[74]. Majority of the deaths are caused by snakes of genera - Naja (cobras), Bangarus (krait), Vipera (Viper). In India only cobra bites cause more anually 20000 deaths every year.

Snakebite is common among adult male and children either due to carelessness or curiosity. Ethnic factors increase the incidence. Males are bitten more than females - 4:1, age 10-19 years,[4]

DETERMINANTS OF SNAKEBITE INCIDENCE :[19]

l) Frequency of contact between snakes and humans depend upon.

- Population density.
- Diurnal and seasonal variations in activity
- Types of behavior (eg : Human agricultural activities).

2) Snakes irritability-Readiness to strike when alarmed or provoked - varies with species.

DENSITY OF SNAKE POPULATION :

Population of snakes varies in geographical area depending upon climate, breeding habits and vegetation. Warmer tropical climate and tropical vegetation are natural environment of snakes. Highly poisonous snakes like king cobra are found in deep forests and snakebites are encountered occasionally.

A snake of moderate toxicity like Echis carinatus is dreaded as the most poisonous snake because of its high population.

In Indian subcontinent Russell's viper population is quite high and the snake is aggressive as well.[23]

LOCATION :

Nearly 75% of snakebites are reported from out door areas. 88.6% of snakebites are reported from rural or semiurban areas.[23]

DIURNAL VARIATION :

More than 80% of snakebites occur between early morning to midnight coresponding to maximum outdoor activity. The nocturnal bites out number the diurual bites, as snakes are predominantly nocturnal in habitat. [1,57]

SEASONAL VARIATION :

In India majority of snake bites occur in rainy season (between May and September) the rain fall fills burrows and drives the snakes away from the burrows out of their resting places. [4,21] Another small peak is seen during March and April.[21]

AGE AND SEX :

Although snakebites is observed in all sex groups the majority of the victims are found in the age group of 11 - 50 years, approximately around 71%. The incidence is more in male population because of their high out door activity as compared to females.[52]

SITE OF BITE :

Snake bites are common in extremities more so in the lower extremities mostly on the foot and in the upper extremities in the fingers, suggesting most bites are accidental [4]

O**CCUPATION :**

Certain occupational groups are vulnerable to snake bites. They are agricultural workers, fishermen, snake charmers, herpetologists and zoo workers. Rural dwellers like farmers and land workers come in close proximity to snakes[67].

SNAKES

Evolution of snake dates back to 70-80 million years during Cretaceous period. They belong to order Squamata, suborder serpents and infra order caenophidae.

Snakes are cold blooded reptiles. Anatomically snakes have elongated head, trunk and tail. The body is covered with scales. Eyes have round or vertical pupils, but lack eyelids. The range of vision is limited. They have primitive internal ears, but no external appendages. In poisonous snakes the teeth modified into fangs which have channels to pour the venom. They have elementary digestive, respiratory, urogenital, circulatory and nervous system.[23]

Of the 2500 - 3000 species of snakes, about 500 belong to the four families of venomous snakes, Tyractaspididae, Elapidae, Hydroplilidae and vipiridae. Only about 200 species have caused death or permanent disability by biting humans.

More than 30 species of another family, Columbridae once considered harmless, have produced signs of envenoming in man and five species have caused fatal bites. Among envenomous snakes only the giant constrictor, Boidae is potentially dangerous to man. The poisonous snakes found in India belong to families Elapidae and Vipiridae [59] About 216 species are found in india, out of which 52 are poisonous. Commonly encountered poisonous snakes in india are cobras, kraits, vipers, saw scaled viper and Russell's viper.[30]

There are few non - poisonous snakes of medical importance are rat snake, water snake, Indian egg eater…,

Medically important snakes species have one or more pairs of teeth with grooves or venom channels, the fangs, in the upper jaw, by which the venom is introduced through the skin of the human victim. Adders posses very long front fangs on which

they impale their victims by a side swiping motion, the fang protruding from the corner of partially closed mouth.

There is no simple and reliable method of distinguishing venomous from non venomous snakes[12]. The mouth can be examined for the presence of fangs but these may be very small in the case of elapids, and folded back inside a sheath in vipers. The characteristic hood of cobras and other elapids is evident only when the snake is rearing up in a defensive attitude. Vipers are often identifiable by their repeated and sometimes colorful dorsal pattern. Russell's viper and puff adders make a loud hissing sound by expelling air through their large nostrils.

The saw scaled or carpet vipers produce a characteristic rasping sound by rubbing their coils together. Rattlesnakes produce an unmistakable sound[19].

IMPORTANT VENOMOUS SNAKES OF THE WORLD: [10]

VIPERIDAE-RUSSEL'S VIPERS, CARPET VIPER ,PIT VIPER, RATTLE SNAKE.

ELAPIDAE- COBRAS, KRAITS, MAMBAS, CORAL SNAKES,

HYDROPHIIDAE (SEA SNAKES)- ENHYDRINA SCHISTOSA, HYDROPHIS CYANOCINCTUS, LAPEMIS HARDWICKII, PELAMIS PLATURUS, AUSTRALIAN LAND SNAKES,

COLUBRIDAE-DISPHOLIDUS TYPUS (BOOMSLANG), THELOTORNIS KIRTLANDI (BIRD SNAKE)

SPECIES OF SNAKE PROBABLY RESPONSIBLE FOR MOST DEATHS AND MORBIDITY IN INDIA :[19]

1) Naja naja- Asian cobras.
2) Bungarus - kraits
3) Daboia russelii - Russell's viper.
4) Colloselasma rhodostoma - Malayan pit viper.
5) Trimersurus - green viper.
6) Echis carinatus, Echis sochureki - saw scaled or carpet viper.

Particulars	Cobra	King cobra	Krait	Viper	Echis
Habitat	Forest dweller	Forest dweller	In plain and cultivated fields	Roaming cultivated fields, bushy & grassy spots	Open area, dry & rocky desert.
Characteristics	Hooded, truncate frontal shield with 3 small post ocular scales & 7 supra labial of which 3rd is largest	Hood frontal shield is not truncated & 2 large occipital shields seen beyond parietal scales	Quite & inoffensive small mouth & fine fangs. Central row of hexagonal dorsal scales prominent on tail	A big slumpy snake, be pit, scales, gigantic fangs, loud hiss, nocturnal, aggressive, good fighter	Gregarious, small stumpy, strikes without provocation, when excited shape of 8, peculiar sound due to rubbing of the folds 8 - 12 mg
Venoms/ Bite	200 mg	200 mg	5-10 mg	150-200 mg	8 - 12 mg
Envenoming	Holds the object of bite and may remain attached to the person	Holds the object of bite and may remain attached to the person		Quickly inject the *venom and* to speed away	
Identification	Usually identified by the person separated after forceful movement	Usually idenditified by the person separated after forceful movement		Not identified by the person	
Fatal dose	10 - 20 mg dry	10 - 20 mg dry	2mg dry	50 mg	50 mg

DESCRSIPTION OF SNAKE SPECIES :

1. VIPIRIDAE :

Vipiridae have long erectile fangs. They are subdivided into two types. Vipiridae and Crotalidae. The venom of both of these snakes is vasculotoxic[39].

Head is triangular with a narrow neck. The scales on the body and the neck are small and of uniform size. They have larger fangs, which are tunneled, and bite marks are more prominent. Often the snake hangs on to the limbs and has to be disentangled by violent movements . Pupils are vertical [55].

RUSSELL'S VIPER : Vipera suselli:

They often grow upto lm and shows 3 rows of oval rings on the body, running along the whole length [25] .It has triangular head with elliptical eyes [33]. The body color is brown. It is robust and idle. It makes loud hissing noise, which at heard over 25 feet. Fangs are very big up to 16mm. There are 2 fangs with 5-6 reserve fangs [65]. Head scales are small and the head is triangular. Dorsolaterally on the body there are diamond or circular shaped spots [59].Head of the russell's viper has the mark of the letter "V" with the apex in front and the body has 3 rows of large black **spots.** The rows **resemble chains** (Chained viper)[55].

SAW *SCALED VIPER* : Echis carinatus :

They are small snakes of 60cm. They are distributed in Africa, West India, Srilanka. It is greyish brown in color with a series of pale spots all over the body**.** They have overlapping scales covering its body and a broad arrow mark on its head and two rows of wavy bands running longitudinally

PIT VIPER: Trimeresurus gramineus :

These are found only in Himalayan belts in India. Pit vipers belong to the crotalidae and are less common in India. They show a depression between the nose and the eye, the loreal pit[25]. Head scales are large and a pit is present between eyes and nasal shield [59].

Pit-viper venoms, containing as many as 15 enzymatic components and an undetermined number of nonenzymatic, low molecular - weight polypeptides, are among the most complex venoms known. The major enzymes in pit viper venoms hyaluronidase, phospholiapse A and various proteinases[38].

ELAPIDAE:

This family includes cobras, kraits, mambas, coral snakes, tiapans, tiger snakes. All have oblong head with well - developed head shields and elongated tails.

COMMON INDIAN COBRA : Naja naja naja :

Head scales are large, the third supralabial scale touches nasal and eye shields. Between the 4^{th} and 5^{th} infralabial scales the wedge shaped scale called cuneate is present. When living it has a hood with or without a spectacle mark. On the under surface of the neck 2 or 3 black bands are seen [59] It shows a monocellate or binocellate dark ring on the dorsum. When provoked the head and reck are raised to form the. hood and might not be evident after it is killed [55]. The fangs are small and anteriorly grooved[25] . The shields under the tail are double. The snake can attain a length of 1.8m. The height to which it can is 60 cm.

It is commonly found in India and Srilanka, except in northeast regions, are most abundant in Orissa, West Bengal, Bihar, Andhrapradesh, tamilnadu, maharastra and Gujarath [10].

It prefers flat grasslands. It shelters in holes of white ant hills, bade of trees and rat holes. It is diurnal hunter.

KING COBRA (Naja hanna) (Ophiophagus ***Hannah)***

They grow to large sizes, often 3 - 4m. They are the world's largest venomous snakes, the largest specimen was 5.71m long and 12 kg weight. It is found in dense forests of India, in Western Ghats, Orissa, Bengal and Assam. It is diurnal in habitat.

SPITTING COBRA :

Spitting cobras can eject their venom under pressure aimed at the eyes and cause intense chemical conjunctivitis with pain, blepharospasm, palpebral edema and alucorrhea[13]. The venom may erode the cornea and can be absorbed into the anterior chamber of the eye causing hypopyon and anterior uveitis. Sequelae include panophthalmitis or corneal opacities [13].

Monocled cobra : Naja najakauthia:

It is more virulent and aggressive than common cobra. It has distinct monocellate circular mark of cream, yellow or orange hood surrounded by a black border. It is found in West Bengal, Orissa, Bihar, Burma and South China, it is an exellent swimmer.

NAJA naja oxiana

It is black in color, hood is not so wide(23).

Common krait (Bungarus caeruleas)

It is a small snake. Average length is 1 metre. Color is blue with paired crossbars. Head scales are large. There are white cross lines seen from neck to tail.

VENOM

During bite fangs of the snake bypass the primary barrier, the skin, deposits venom in blood vessels, peritoneum, subcutaneous tissue or muscle. Absorption of venom is variable, but it takes place most rapidly through the blood vessels. In subcutaneous tissues the venom is transported to the other sites via the lymph channels and capillaries [39.]

Hyaluronic acid and phospholipase - A play a role in local diffusion of the venom and penetration into lymphatics. Viper venom enters circulation slowly and produces systemic effect as early as 12 hr. and maintains coagulation disturbances for weeks.

Distribution of venom depends upon protein binding, membrane permeability, and pH, with entry into tissue based on the rate of blood flow and tissue mass. Upon reaching these tissues, venom crosses the membrane by four methods of transport: passive diffusion, facilitated diffusion, active transport and pinocytosis [(4S)].

Severity of the envenoming depends upon the following factors :

1) Dose of venom injected - depends on mechanical efficiency of the bite, species and size of the snake,

2) Composition and hence potency of venom - depends on species and, within a species, the geographical location and age of the snake

ENZYMES

The snake venom contains several enzymes (26) enzymes but no snake contains all of them.

- **Proteolytic enzymes : Proteinases** :

Snake venom contains trypsin like enzymes to digest tissue protein and peptides, which are inactivated by EOT A due to chelation of the catalyst zinc. Venoms are rich

in protease activity and produce marked tissue destruction, hemorrhagic activity. **Proteinases** are **abundant in viper and cratolid** venoms.

- Arginine easter ***hydrolase*** :

  It induces bradykinin activity and affects clotting

- **Collagenase** : Digests collagen.

  Hyaluronidase : **Catalyses cleavage of internal** glycoside bond resulting in connective tissue viscosity facilitating the penetration of the venom, it hydrolyses hayaluronic acid.

  Its net effect is to cause (1) an increase in cell wall permeability with hydrolysis, disruption and alteration of connective tissue allowing further spread of toxin (2) muscle and subcutaneous tissue damage leading to necrosis (3) promotion of intravascular clotting and fibrinolysis resulting in defibrination syndrome [39] .

  Phospholipase A: Catalyses hydrolysis of fatty ester linkage resulting in release of both saturated and unsaturated fatty acids causes uncoupling of phosphorylation and inhibits respiration, causes hemolysis due to lysophosphatidase [39]. It can inhibit the electron transfer at the level of cytochrome C and solubalize mitochondrial bound enzymes. It can hydrolyze the phospholipids on the nerve axons, breakdown the acetylcholine vesicles at the myoneuronal junctions, cause myoneuronolysis and lyse RBC cell membranes, by acting on the phospholipids thus causing the hemolytic effects [24]. Thus it has synergistic effects on cardiotoxin and neurotoxin activity.

  Phospholipase A2 : most widespread and extensively studied of all snake venoms. It damages mitochondria, RBCs, WBCs, platelets, skeletal muscle, vascular endothelium. It produces presynaptic neurotoxic activity, opiate like sedative effects and auto pharmacological release of histamine[(19).]

  Phospholipase B*:* Hydrolysis of lysophosphatides.

***P*hosphodiesterase**: Attack on DNA,RNA, and arabinase derivatives.

Acetyl cholinesterase: Causes hydrolysis of acetylcholine to acetic acid and choline. It is an important enzyme of elapid venoms. It has been claimed to have curare type of effect and thus claim to neurotoxicity.

5 Nucleotidase: Causes hydrolysis of mono esterase linked with 5 positions of DNA and RNA.

L -amino acid oxidase: Almost all snakes have this, which catalyses the oxidation of L – alpha aminoacid and alpha hydroxy acids.

Phosphatases: Present predominantly in elapid venom. it causes profound fall in blood pressure.

POLYPERPTIDS :

Venom contains low molecular weight proteins without enzymatic activity

Cobrotoxin : cardiotoxic, hemotoxic, anticoagulant and neuromuscular disrupt. Closely homologous to neurotoxin and causes depolarization of membranes resulting in neuromuscular blockade, axonal conduction blockade, hemolysis, muscular contraction and cardiac arrest [19]

Ceruleotoxin : Indian krait contains ceruleotoxin, a highly lethal neurotoxin that acts on the postsynaptic site without binding to Ach receptor.

Hemorrhagins : They exert their main toxic effect on the lungs and distrupt endothelial cell junctions and alveolar septa, which results in increased hemorrhage into pulmonary arterial and arteriolar walls, intraalveolar movement of blood and transudates, and eventually, pulmonary congestion and increased lung weight [8,48]. These effects can also occur locally at the bite site, causing hemonhagic edema and systemic bleeding leading to shock [39].

Neurotoxins: Neurotoxins can be found in elapid, hydropid, vipirid and cratolid

venoms. They are neuromuscular non depolarising blocking agents with different methods of action [39].

VENOM TOXINS CAUSING HEMATOLOGICAL EFFECTS

Procoagulant activity: Snake venom acts as procoagulant and anticoagulant by interacting at all six sites of coagulation.

- Factor X activation: Venom activates factor X due to its Co dependable nature, as calcium binds to gamma glutamic acid residue in factor X.
- Factor IX activation: By causing cleavage of single peptide bond, which is enhanced by the presence of calcium ions.
- Factor V activation: Increases the activity of Factor V by decreasing the molecular weight of factor V.
- Direct prothrombin activation: Venoms of elapids, vlpers and colubrids have this property, which can be inhibited by EDTA.
- Thrombin like action: Venoms of Crotalidae and viperidae constitute significant amount of thrombin like enzyme which are mostly glycoprotein and serine esterase which are not inhibited by heparin, protease inhibitors. They act in several ways:

1. Remove fibrinopeptide A
2. Never activates factor XIII.
3. Additional fibrinogen degradation.
4. Lack of platelet aggravating activity.
5. Act as coagulant in both vitro and Vivo.

- Anticoagulant activity: inhibits one or more of blood clotting factors or prevent activation of one of the clotting proteins.

1) Have fibrinolytic or fibrinogenolytic action by direct action on fibrin or fibrinogen.

2) Activate by fibrinolytic mechanism by direct action on plasminogen.
3) Inhibit clotting by direct action of the venom's anticoagulant with phospholipids.

Russell's viper venom contains atleast two proteases, which activate the mammalian blood clotting cascade. RVV - X, a glycoprotein, activates factor X by a calcium dependent reaction and also acts on factor IX and protein C. RVV - V, an arginine ester hydrolase, activates V (19).

- Echarin: Echis venoms contain a Zinc mettaloprotein, Echarin, which activates prothrombin (19).
- Ancrod (Arvin): Malayan pit viper venom contains a glycoprotein serine peptidase, ancrod (Arvin) which cleaves fibrinopeptide A, from the fibrinogen molecule (19).

COAGULATION DISORDERS IN POISONOUS SNAKE BITES

Hematological complications are seen after the bite of some Vipers, pit vipers, elapids and colubrids. The hematotoxic effects of snake venom is a summation of three possible enzymatic mechanisms of varying degree:

1. Coagulation
2. anticoagulation
3. fibrinolysis [30]

Venom procoagulants can activate intravascular coagulation and produce consumption coagulopathy leading to in coagulable blood. Procoagulants in the venom of Colubridae, Echis and Notechis scutatus activate prothrombin. Daboia ruselii venom has procoagulants activating factors V and X. Many Cratolinae venoms have a direct thrombin like action in fibrinogen. Venoms of rattlesnakes cause defibrinogenation by endogenous fibrinolytic system [(33).]

Thrombocytopenia is a common accompaniment of systemic envenoming.

Spontaneous systemic bleeding is attributable to distinct venom component, haemorrhagins, which damage vascular endothelium. The combination of defibrination, thrombocytopenia and vessel damage will result in massive bleeding, a common cause of death from viper bite.

Two immunologically non – enzymatic hemorrhagic principles- haemorrhagins 1 and 2 are typical components of cratolid and viperid venoms. They act by disrupting the endothelial lining and by inhibiting platelet aggregation. Pharmacological studies have shown that they induce the release of histamine and 5 - HT which may mediate hemorrhagic effects [47].

BLOOD COAGULATION :

Most venom exhibits anticoagulant effects: It is essentially due to hypofibrinogenemia brought about by massive intravascular fibrin clotting. Russell's Viper venom is rich in enzymes that activate factor X to convert prothrombin to thrombin in the presence of calcium, factor V and platelets. A thrombin like action is almost restricted to cratolid venoms and accounts for marked coagulant effects without accompanying thrombocytopenia, hypocalcemia or reduction in factor V activity in the plasma [29].

INTRAVASCULAR HEMOLYSIS [19] :

Envenoming by Russell's viper may be associated with massive intravascular hemolysis. Evidence of mild microangiopathic hemolysis is sometimes found in patients with severe bleeding and clotting disturbances.

COMPLEMENT ACTIVATION [19] :

Elapid and Colubrid venoms activate complement via the alternate pathway where as viper venoms activate via classical pathway. Pituitary hemorrhage has been described.

DIRECT LYTIC FACTOR:

This is a polypeptide of molecular weight of 2000, lyses washed RBCs. It is produced by few

cratolid, viperid and elapid snakes. By itself it is weakly hemolytic, but when direct lytic factor and phospholipase A are both present strong hemolytic action can occur.

CLINICAL PRESENTATION

Clinical presentation of snakebite may be straightforward and obvious, but not always. The history may be vague and confusing and the physical findings minimal or absent. Thus a hypotensive, unconscious adult or a child with acute seizures, but no obvious clue of the cause [39] may challenge the clinician. The earliest systemic symptoms such as vomiting may develop within minutes of the bite [19].

Clinical presentation in snake bite depends on the fol1owing factors:

1. Age and general health of the patient.
2. Nature, location, depth and number of bites.
3. Amount of venom injected.
4. Conditions of fangs and venom glands.
5. Victim's sensitivity to venom.
6. Pathogen present on snake's mouth.
7. Degree and kind of first care.

LOCAL PRESENTATION:

PAIN:

Local pain is the most common symptom. However it is most severe in bites of vipers and cobra. Most of the severe pain results secondary to intense local swelling, painful lymphadenopathy. The pain usually manifests within 30min [39] .

BITE MARK: The *usual* number of punctures is two; however, there can be one or three marks present because a fang could have been lost or a replacement fang may be present without the older one having been shed.[39]

Coral snake's puncture marks are usually separated by a distance of 2 - 8 mm [39] the fang marks may be quite small and difficult to identify [38]. To complicate matters, sometimes no visible marks can be found [38].

LOCAL SWELLING :

In the bitten limb increased vascular permeability leads to swelling and bruising. The factors responsible include proteases, phospholipases, membrane damaging polypeptide toxins, hyaluronidase and endogenous autocoids released by the venom, such as histamine, 5-HT, kinins [19].

Local swelling is evident within 2 - 4 hrs of bites by vipers and cytotoxic cobras and may develop even more rapidly following bites some rattle snakes.

With cratolid evenomation local pain and swelling are noted most frequently and progress rapidly up the extremities, particularly within the first hour, then more slowly over the next few hours. Within first 30 min the patient may complain of weakness or numbness and tingling of the face and lips. Skin discoloration can be noted early with associated regional Iymph node pain and swelling. With severe evenomation or inadequate treatment, the signs and symptoms can progress rapidly with petechiae, ecchymosis, bleb formation and subsequent necrosis [39]. Swelling is maximal and most extensive on 2nd or 3rd day after the bite. Resolution of swelling and restoration of the normal function in the bitten limb may be delayed for months especially in older age [19]. A corel snake bite may how no early signs of evenomation, although subsequent progression can be rapid pain. Local swelling may be minimal [39]

During initial examination several locations on bitten extremity should be marked and the circumference is measured. Measurements should be repeated every 15 min until swelling is no longer progressing and every 1- 4 hrs thereafter. [38] Limb circumference should be noticed at several sites above and below the bite, and the advancing border

of edema should be outlined every 15 min [40]

TISSUE NECROSIS:

Local tissue necrosis results from the direct action of myotoxic and cytolytic factors, possibly polypeptide toxins, and ischemia caused by thrombosis, tourniquet and compression of arteries by swollen muscle within the fascial compartment [19.] Dusky discoloration is noticed around the bite mark which extends in areas and deepens in color each day. About 3rd or 4th day it is encircled by a red rim with small blisters. Then later necrosis develops rapidly within a few days. Sloughing of the necrotic tissue and secondary infection including osteomyelitis may occur during subsequent weeks or months[34]

LYMPHADENOPATHY:

It is common in viper bites. It is a clue to the fact that venom is absorbed through lymphatics [58]

SYSTEMIC PRESENTATION:

Any of the following system can be involved by the envenomation depending on the composition of the venom:

1. Hematotoxicity.
2. Neurotoxicity.
3. Cardiotoxicity.
4. Renal failure.
5. Myotoxicity.

NEUROTOXICITY:

It is seen predominantly with Cobra and Krait [56]. Within 20 minutes to 15 hours of bite it leads to bilateral ptosis and ophthalmoplegia. Other complications are dysphagia,

respiratory muscle failure, cyanosis, drowsiness, delirium and coma [67]

CARDIOTOXICITY :

It is commonly seen with Elapidae and sometimes with Viperine bites [75].

Symptoms of hypotension, cardiac anythmias, cardiac arrest, peripheral circulatory failure, myocarditis and pulmonary edema appears within 30 minutes to 2 hours [31.] Peripheral circulatory failure seen with viper bites are mainly due to hypovolemia.

RENAL FAILURE:

It is seen mainly with viper bites intravascular, haemoglobulinutria, myoglobulinuria, shock, DIC, fibrinolysis brings about renal damage [61]. Renal changes include glomerulonephritis, vasculitis, cortiical necrosis, renal infarction, interstitial nephritis and acute tubular necrosis [35.]

MYOTOXICITY: Snake bites are associated with degeneration, necrosis of muscles, myoglobinuria and raised potassium [35]

HYPOTENSION AND SHOCK: Profound hypotension is a part of autopharmacological syndrome, which may occur within minutes of bites by Vipers. Presumably this is caused by release of vasodilating autocoids. Oligopeptides in viper venom inhibit bradykinin -deactivating enzymes and ACEs [19]

CLINICAL FEATURES IN SPECIFIC SPECIES OF SNAKEBITES:

VIPERIDAE: Pain, edema, swelling, ecchymosis, vesiculation, hemorrhagic bullae, petechiae, necrosis, shock, consumption coagulopathy, bleeding, prolonged clotting, fall in platelet count.

The envenomated patient typically experiences moderate to severe pain at the bite site

within 5 to 10 minutes [37]. The pain is often described as burning and may radiate along the bitten extremity. Swelling at the bite site soon follows and may progress along the entire extremity within hours. There is often local ecchymosis due to disruption of blood vessels. Since the venom is largely absorbed via the lymphatics, impressive lymphangitis may appear early[38].

There may be increase or decrease in heart rate, and low blood pressure.

Early shock is due to pooling of blood in pulmonary and splanchnic vascular beds or in the bitten extremity. Delayed shock is due to hypovolemia from bleeding or bleeding into the third space and hemolysis. Pulmonary edema is due to destruction of intimal lining of pulmonary blood vessels. Renal failure is not uncommon. Occasionally paresis or even frank paralysis is seen [38].

Venoms of viper and pit viper usually produce more local effects than other snake venoms. Local blistering appears after 12 - 24 hrs of bite and may extend up the limbs(13). Blistering may appear at the bite site as early as 12 hr. after the bite and may be filled with clear or blood stained fluid. Necrosis of the skin, subcutaneous tissue and muscle may develop in up to 10% of the cases. Bites on digits and tight fascial compartments particularly likely to cause necrosis. Sudden severe pain, absence of arterial pulses and a demarcated cold segment of the limb suggest thrombosis of the major artery. The absence of the detectable local swelling 2 hr. after the viper bite usually means no venom was injected. But there are exceptions to the rule. Fatal systemic envenoming by rattlesnake and Russell's viper may occur in the absence of local signs. [19]

Persistent bleeding from the fang marks and from new injuries such as venepuncture and from partially healed wounds may be the first indication of bleeding diasthesis [72].

Spontaneous systemic hemorrhage is most often selected in gingival sulci. Saliva and

sputum may contain blood, which usually derives from bleeding gums or epistaxis. True hemoptysis is rare. Hematuria can be detected a few **hrs** after the bite. Eccymosis, intracranial and subconjunctival bleeds, and bleeding into the floor of the mouth, tympanic membrane, and GI and genitourinary tracts also occur. Discoid ecchymosis, especially of the face and trunk are seen in bites of Russell's viper. Bleeding into anterior pituitary has been described by the bites of Indian Russell's vipers. Menorrhagia, antepartum and postpartum hemorrhage have been described in women bitten by vipers. Severe headache and meningism suggest SAH [26]. Hemiplegia, irritability, loss of consciousness and convulsions suggest an intracranial hemorrhage. Abdominal distention, tenderness and peritonism with signs of hemorrhagic shock suggest retroperitoneal or intraperitoneal hemorrhage. Intravascular hemolysis causing hemoglobinemia and black urine has been described in patients bitten by SriLankan Russell's viper [19]

Conjunctival edema will develop as well as serous effusion, pulmonary edema, hemoconcentration and a fall in the serum albumin[19]

PIT VIPER ENVENOMATION:

The earliest and almost diagnostic symptom of hematotoxicity is the appearance of hemorrhagic blebs with bleeding from the site of bite. Within 2 - 48 hr of bite patients may present with varying degree of hemorrhagic manifestations. Frank or microscopic hematuria has been observed to be the commonest symptom.

Other rare bleeding sites are subconjunctival bleed. Hemoptysis has been reported as the commonest bleeding manifestation in Malaysia. In Indian studies hematuria is the commonest bleeding *problem* [1.4,50]. Death is usually due to shock or hemorrhage.

ELAPIDAE:

50 - 60% of patients bitten by elapids will not develop local or systemic effect.

Local: Pain, local swelling, local necrosis, vomiting, headache, syncope, abdominal pam, drowsiness, pallor, sweating.

Systemic: Vasomotor signs - weak pulse, hypotension.

Neurotoxicity -,bilateral ptosis, respiratory failure, bulbar palsy, muscular palsy.

Elapid venoms are best known for their neurotoxic effects, which may develop as soon as 15 - 20 min after the bite, or after several hours [23.] In some cases local effects are minimal. Local swelling may develop as well as regional lymphadenopathy. Blistering may appear within 24 hrs. Secondary infection can occur [19.] Chronic ulceration may develop with resulting Squamous cell carcinoma years later [13.]

Generalized myalgia and peripheral motor neuropathy in some victims of kraits and corel snakes probably result from the damage to the muscle and nerve terminals by phospholipase A2 [13]

Preparalytic symptoms include vomiting, blurred Vision, paresthesia, hypersalivation, conjunctival congestion and gooseflesh.[13] Neurotoxic Signs progress in the following order; ptosis, paralysis of upward gaze, total external ophthalmoplegia, inability to open the mouth, protrude the tongue, speak and swallow, respiratory paralysis and generalized flaccid paralysis [13] . Elapids can cause persistent bleeding from the wounds and spontaneous systemic bleeding from the gums and GI bleeding. [19]

Severe envenoming by the king cobra results in swelling of the whole limb and formation of the bullae at the site of the bite. But there is minimal local necrosis. Rapidly developing neurotoxicity is a dominant clinical feature. The earliest symptom of systemic envenoming is repeated vomiting. Neurological signs may appear as early as 15 min, but may be delayed for 10 hrs. Many of the patients are unable to open their

mouth. Respiratory paralysis results, which leads to respiratory failure and hypoxemia, which in turn leads to unconsciousness and convulsions [19].

HYDROPHIDAE:

Successful envenomation is seen in 33% of patients. The bite is usually painless and may not be noticed by the wader or swimmer, but the teeth are frequently left in the wound. There is no local swelling or involvement of local lymphadenopathy. Mainly myotoxic, caused dysphagia, ophthalmoplegia, myoglobinuria, renal failure. Generalized rhabdomyolysis is the dominent feature. Early symptoms include headache, thirst, sweating, vomiting. Generalized aching, stiffness and tenderness of the muscles are seen between 30 min to 4 hrs. Later there is progressive flaccid paralysis starting with ptosis. Myoglobulinurea results [19].

COLUBRIDAE:

All the species give similar symptoms. Nausea with repeated vomiting,Colicky abdominal pain and headache develop hours after bite. There may be bleeding from old and recent wounds, spontaneous gingival bleeding, epistaxis, hematemesis, melena, SAH, hematuria and extensive ecchymosis. Intravascular hemolysis and microangiopathic hemolysis has also been described. Most of the cases die of renal failure from acute tubular necrosis. Venom procoagulant activates prothrombin which triggers DIC leading to elevated FDPs, severe thrombocytopenia and anaemia [19].

ATRACTA SPIDIDAE:

Local effects include pain, swelling, blistering, necrosis, tender enlargement of local lymph nodes, paresthesia. The most common symptom is fever. Most of the cases die within 45 min of the bite after vomiting, producing profuse saliva and lapsing to coma. Rarely mild abnormalities of blood coagulation have been described [19].

FIRST AID

Reassurance is vital, for the bite does not always mean that envenomation has occurred [11,9.]

First aid can be carried out by the person who is bitten or by others who happen to be nearby at the time, using materials which are readily available close to hand.

1. Reassure victim, who will almost certainly be terrified.
2. Put the victim at rest.
3. Do not hamper with the bite wound in any way, but immobilize limb using a splint or sling. If available firm binding of the splint with a crepe bandage is a effective form of immobilization.
4. Application of constriction band, incision and suction.
5. Take the patient to nearest health clinic or hospital. Muscular contraction in the bitten limb will promote spread of venom.
6. Avoid harmful and time wasting treatments.
7. The snake should be taken to hospital.[10]

Don't include cauterization, excision, amputation, mouth suction, chemical instillation, ice cooling and electric shocks [19.]

CONSTRICTION BANDS:

Must be wide and should be applied 2 - 4 inch above the bite to occlude the lymph flow. Band should not be applied around the neck, joint, trunk.[16,9.] In pit viper envenomations, the most commonly accepted first aid measure is application of a wide (> 1/2 inch) constricting band 2 - 4 inches above the bite site, only tight enough to occlude the superficial veins and lymphatics. If applied within 30 minute of bite this may limit some central spread of venom [40.] The use of tourniquets, compression pads

and bandages remains controversial. In animals it is proved effective in preventing the venom spread and prolongs the survival [46,9.] Dangers of tourniquets and other occlusive methods include ischemia and gangrene, if they are applied for more than about 2 hrs, damage to peripheral nerves, increased fibrinolytic activity, congestion, swelling, increased local effects of venom and shock or rapid development of life threatening systemic envenoming after their release [10.] A tourniquet applied around upper limb, must be tight enough to obliterate the peripheral arterial pulse. This should be released for about 30s every 30 min. Alternately if crepe bandage is available, the limb can be firmly bound to a splint, which is far less uncomfortable and dangerous and may be as effective.

INCISION AND SUCTION:

The method of incision and suction associated in the past has been a source of controversy because in delaying 23% of labeled venom was removed after 3 min and 34% was removed after 30min.[7] The scientific jury is still out on this device, but it is promising and therefore incision and suction of the bite have been recommended as first aid measures. The incision should be parallel to the axis of the extremity and should be only approximately 6mm long and 3mm deep. 1t should not be more than 3cm long and 3cm deep [16.] Ice should be avoided [38.] Preferably done by suction cup. If done by mouth venom should not be swallowed by mouth.

ICE AND COOLING: ICE should be avoided. The American red cross society recommends that no cooling measures of any kind be used in venomous snakebites. *Cooling* may actually drive some venom component deeper in the tissue. Cryotherapy, popularized in 1950's consisted of packing the bitten extremity in ice water for 6 - 24 hr,or longer. This technique resulted in large number of amputations and was subsequently abandoned. [38]

ELECTRIC SHOCKS *:*

Controlled animal studies have failed to demonstrate the efficacy of the high voltage, low ampere electric shock technique that is practiced in Ecuador.[38]

IMMOBILIZATION AND SPLINTING:

Immobilization and splinting of extremity at approximately at heart level and then transporting the victim as soon as possible to the hospital are extremely important.[38]

INVESTIGATIONS

When the patient arrives to emergency room, the following basic studies should be done. Complete blood count, platelet count, coagulation studies (PT, aPTT, fibrinogen level and fibrin degradation products - FDRs, electrolytes, creatinine, BUN levels, blood group and cross matching [ll]. Lab studies should include the following: blood glucose, LFT, amylase, CPK, arterial blood gases, urine analysis, urine myoglobin [38.]

Hematocrit may be high initially because of hemoconcentration where there is generalized increase in capillary permeability. A subsequent fall in hematocrit is usual. The causes include bleeding into the bitten limbs and elsewhere and uncommonly, from intravascular hemolysis, especially microangiopathic hemolysis in patients with DIC. Most species excites a neutrophilic leucocytosis counts above 20000 cells/microliter indicate severe poisoning.

HEMOGLOBIN: A mean fall of 3g% has been noted In one study. 50% of victims had a Hb% of 9.3% or less.[4]

W. B. C COUNT : Neutophilic leucocytosis has been described as one of the features of envenomation in some series. In other series a high leucocytosis has been observed only in cases where local necrosis has occured due to infection [70.] Neutrophil count more than 20000 cells / cubic mm indicates severe envenomation. [71]

RED CELL MORPHOLOGY :

It is rare to find evidence of microangniopathic hemolysis with numerous schizocytes, poikilocytes and thrombocytopenia. Some patients may show some evidence of microangiopathic hemolysis with slight sphering of red cells and red cells fragmentation. Spherocytosis, acanthocytosis and Heinz body formation have been described and attributed to a direct toxic effect of venom on R.B.C membrane[5]

PLATELETS COUNT:

In Russell's viper bite the platelet count may be low [19]. Platelet count is an important component in the evaluation of hemostasis. It can be done by manual or by automated techniques. [4,50] Platelet count are best performed on EDT A anticoagulated blood normal platelet counts are performed by visual examination of diluted whole blood. Automated methods are based on aperture (or) electronic impedance (or) light scattering technology.

BLEEDING TIME:

Normal value is 2 to 4 minutes [17]. Lack of clotting factors leads to prolongation of BT, especially lack off platelets. Approximately 30% of patients bitten by vipers have prolonged bleeding time and in 25% Hess test is positive[(4,50).] Bleeding time is a screening test for detecting disorders of platelet function and is directly by platelet count and the ability of platelets to form a plug [22,29]

DUKES METHOD:

It is one of the commonly used method. In this a prick is made and the filter paper is used every half minute, until bleeding stops. The time from the prick to the stoppage of the bleeding is the bleeding time [6.]

IVY'S METHOD:

It is an improvement over Dukes. Here a uniform pressure is applied on blood vessels

by blood pressure cuf inflated to 40mm Hg and two standardized punctures on forearm are made using disposable lancets. The blood is mopped every 30 sec by a blotting paper. Normal range 2 - 7 Mins.

WHOLE BLOOD COAGULATION TIME:

CT involves factors involved in the intrinsic pathway for clotting.

Methods:

1) **Capillary glass tube method**: Blood is collected in a thin capillary test tube and then the tip of the tube is broken every 15 seconds until fine thread of clotted blood appears[6.]

2) **Lee and White method**: 1 ml blood is taken in two test tubes and closed by rubber cork. After 5 minutes, first tube is tilted to 45 degrees and at one minute interval up to 180 degrees without blood flowing out of it. This interval is the clotting time. It is a poor screening test for coagulation activity. Normal time varies from 5 - 15 mins by Lee and White method coagulation time is informative only if it is significantly prolonged. [6]

3) **Wright coagulometer**: Dozen of capillary tubes of equal caliber are fillied with blood, placed in water bath at 37 degree centigrade. After 4 minutes the blood is expelled from the first tube into the water and others at the intervals of 30 seconds. Worm like clot is a positive test[6.]

CLOT RETRACTION:

When the blood coagulation is complete the clot normally undergoes retraction. Clot retraction usually is deficient when platelet count is below 50,000 cells/cmm. It is also a screening test for platelet function.

In hypofibrinogenemia formed clot will be small with increase amount of red blood cell fall out. In DIC the formed clot appear small and ragged and there will be increase

RBC fallout.

PLASMA PROTHROMBIN TIME: (PT)

PT measures total quantity of prothrombin in blood. It was first described by Dr. Quick. The prothrombin time is useful screening procedure for the extrinsic coagulation mechanism including the common pathway. Citrated platelet poor plasma is recalcified in the presence of excess tissue factor and thus "by passes" the intrinsic pathway. Normal range of PT - 12 - 16 sec. Manual and automated methods of clot detection. Blood is oxalated (prevents conversion of prothrombin into thrombin) and later excess of calcium ion and tissue thromboplastin are added. Calcium nullifies oxalate effects and the tissue thromboplastin activates prothrombin - thrombin by extrinsic clotting pathway [6l].

PARTIAL THROMBOPLASTIN TIME:

This is a simple test .for the intrinsic and common pathways of coagulation. It is performed upon citrated platelet poor plasma by activating the contact factors (e.g: Kaolin) adding a standaridized phospholipid preparation as a platelet substitute and then measuring the time for clot formation following the addition of an excess of calcium. The platelet substitutes are only partial thromboplastin and they are incapable of activating the extrinsic pathway, which require complete tissue thromboplastin. Normal range - 28.36 sec. And depends on the reagent used and duration of pre-incubation period [6.]

FIBRIN DEGRADATION PRODUCT

They are protein fragments (X,Y,D,E) of varying sized that results from the proteolytic action of plasmin or fibrin or fibrinogen T 44,45. Normal range <**IO mg/m1**. Three general kinds of assays are available to measures [14.]

I) Staphylococcal - clumping tests.

2)Tanned red cell hemagglutination inhibition assay.

3)Latex agglutination tests.

Staphylococcal clumping tests, employ certain strains of coagulase negative staphylococcus that agglutinate FDP, and measures only fragment X and Y. tanned red cell haemagglutination inhibition test is sensitive to all forms of FDPs but technically more difficult and complex. Latex agglutination assay employs antibodies attached to latex particles to the plasmin - cleaved fibrin fragments recognizes all FDP fragments and are rapid and simple. Other methods of detecting FDP include immunodiffusion and counter immunoelectrophrosis.

D-DIMER:

D-dimer measures plasmin cleaved and factor XIII a - cross lined fibrin *T46. Thrombin cleaves* fibrinogen, *to* soluble fibrin, also it activates factor XIII, which cross links soluble fibrin into *soluble fibrin and the plasma* cleaves *this* cross lined insoluble fibrin, liberating the D-dimer. Detection of D - dimer addresses both thrombin and plasma generation. Normal range - <0.5 microg/ ml. Can be measured by enzyme immuno assay (ELISA) and also by latex agglutination tests, which uses monoclonal antibody coated latex particles against D-dimer neo antigens DD-3B6/22. latex agglutination tests provides rapid and simple test for Ddimer compared to enzyme immuno assay[14.]

COAGULATION DEFECTS:

Incoagulable blood is a cardinal sign of severe poisonmg in snakebites.

This usually *returns to* normal in 24 - 36 hr.[50]. There will be reduction of Factor V,X,X! and XIII. Factor VIII, XI, XIII, protein C and AT - III are also depleted. Fibrin

/ FDP are very high. Plasminogen and antiplasmin levels are reduced. Thus PT, aPTT and thrombin time may be abnormal [50,64].

COMPLEMENT:

C3 may be decreased. C4 and CBG (complement binding globulin) may also be low, suggesting the activation of the complement system by both classical and alternate pathways[9].

URINE ANALYSIS:

1. Oliguria may indicate impending renal failure.
2. Proteinuria is found in the majority of patients who have features of systemic envenomation and appears even before a gross clotting defects detectable. Increasing albuminuria is an indication of glomerular damage, glomerular capillary fibrin deposition and progressive renal damage and failure [35,46,61].
3. RBCs and RBC casts in microscopic examination of urine is an early sign of hematotoxicity.
4. Urine is examined for hemoglobinuria and myoglobulinuria.
5. Radioimmunoassay is done for venom using freeze- dried sample.
6. Renal fractional sodium ions is tested.

BLOOD UREA, CREATININE AND ELECTROLYTES:

In patients with oliguria, the maximum daily rise in blood urea ranged from 3.4 – 21.4 mmol/L to reach the peak of 14-68mmo1/L in 6 days after the bite[64]. Serum potassium and serum sodium should be estimated. Hyperkalemia hyponatremia may be noticed in renal failure patients, but hyperkalemia may also be noticed in rhabdomyolysis and intravascular hemolysis [33].

RBCs led to rise in LDH levels [23.]

ECG

ECG changes are unusual but important. Sinus bradycardia, ST - T changes, various degrees of A V blocks, sinus tachycardia, ischemic changes, tall T waves, short PR intervals to wide range of heart blocks and arrythmias are observed [3,75] and evidence of hyperkelemia have been described especially with viper bite.

CHEST RADIOGRAPH: Chest X rays are useful for detecting pulmonary edema, intrapulmonary hemorrhage and pleural effusion [19.]

EEG: Reduced alpha activity, sharp spikes and diffuse delta activity can be noticed. This suggests encephalopathy. The waves appear within hours of bite and may persists for several days. Changes are seen mainly in temporal lobe.

IMMUNODIAGNOSIS: ELISA was developed to detect the presence of venom at the puncture site or in the patients serum. ELISA is used retrospectively for the detection of envenomation [(60).] Immunological tests are highly sensitive, but the specificity may be inadequate to distinguish between different species and false positive results are common. Relatively high venom antigen concentrations can be detected within 15 -30min, which is just enough to select the appropriate mono specific antivenom [19.]

LUMBAR PUNCTURE :It is indicated. when there is Suspicion of subarchnoid hemorrhage, but C.T can also be done to confirm SAH [2].

MANAGEMENT : The three most important questions in the management of snakebite are:

1)What is the site of bite?

2)What is the time interval ?

3)What kind of snake was it?

If the snake is diagnosed non- venomous the patient can be discharged immediately after receiving a booster dose of Tetanus toxoid [27]. Discovery of 2 -3 fangs marks suggest fangs of a venomous snake. Any patient with suspected venomous should be admitted to an intensive care unit for at least 24 hr [38.]

Management of the patient of snake envenomation should be aimed at:

1. Reassurance of the patient to protect from the death due to fight.
2. Retarding *absorption* of venom by mechanical measure.
3. Venom neutralization by immune serum.
4. Relief of symptoms and prevention of complication [3.]

Local pain may be intense. Oral paracetemol or diclofenac sodium is preferable to aspirin, which commonly causes gastric erosions and could lead to persistent gastric bleeding in incoagulable disorders. Severe pain can be treated with pethidine or pentazocine.[19]

Unless the responsible snake is identified by an expert as non- venomous, all snake bites are' observed in hospitals for 48hrs. level of consiousness, lid retaraction, blood pressure, pulse, breathing, urine output and extent of local swelling should be monitored frequently.[39]

Vomiting is common symptom. Patients should be lie on their with head side with head down to avoid aspiration. Persistent vomiting can be treated with iv

chlorpromazine - 25 - 50 mg in adults and 1 mg/kg in children.[19]

GRADING OF ENVENOMATION:

There are various grading of envenomation described by various authors:

1) Envenomation can be graded as follows: [19]

0 Fang marks without any symptoms and Signs

1. Fang marks and minimal symptoms and signs.
2. Fang marks and moderate symptoms and signs.
3. Fang marks with severe symptoms and signs.
4. Fang marks with acute symptoms and signs.

2) Envenomation can also be graded as follows [73]

ENVENOMATION	REACTION	DOSAGE
None	No local or systemic reactions	No antivenom
Minimal	Local swelling, but no systemic reactions	5 vials
Moderate	Swelling that progresses beyond the site of bite together with a systemic reaction, lab changes or both	10 vials
Severe	Marked local reaction, severe symptoms and lab abnormalities	10-15 vials

ANTI VENOM

Anti venom is a suspension of venom neutralizing antibodies derived from the sera of the hyperimmunized animals using simple ammonium sulfate fractionation[39].

Antivenom is a whole serum or enzyme- refined immunoglobulin of animals, usually horses or sheep, which have been immunized with venom, it is the only specific

treatment available and has proved effective against many of the lethal and damaging effects of venoms [62].

In the management of snake bite the most important clinical decision is whether or not to give antivenom, for only minority of the snake bitten patients need it, it may produce severe reactions and it is expensive and often in short supply [19].

If the biting species is known, appropriate mono - or - poly - specific antivenom should be chosen. If the species is unknown, polyspecific antivenom covering the important species of the region should be used[13]. Most of the antivenoms are polyvalent[39].

INDICATIONS :[43]

SYSTEMIC ENVENOMING :

Hemostatic abnormalities : Spontaneous systemic bleeding, incoagulable blood or prolonged clotting time, elevated FDP, thrombocytopenia.

Cardiovascular abnormalities : hypotension, shock, abnormal ECG, arrythmias, cardiac failure, and pulmonary edema.

Neurotoxicity.

Generalized rhabdomyolysis.

Impaired consciousness.

In patients with definite signs of local envenoming, the following indicate significant systemic envenoming : WBC count more than 20000/ microliter, neutrophilia, elevated serum enzymes such as creatinine phosphokinase and aminotransferase, hemoconcentration, uremia, hypercreatininemia, oliguria, hypoxemia, acidosis and vomiting in the absence of a history of ingesting emetic agents

SEVERE LOCAL ENVENOMING:

1. Any stage of local swelling involving more than half the bitten limb or extensive blistering or bruising.
2. Rapid spread of swelling, even without evidence of systemic envenoming.
3. Tender local lymphnode[19].
4. To improve the rate of recovery of local swelling.
5. Swelling extending up the forearm or leg within 2 hrs of bite.[19]
    a. Known necrotic venom[13]
    b. Bites on digits and into other tight fascial compartments.

CONTRAINDICATIONS :

Atopic patients and those who have had reactions to equine antiserum on previous occasions have increased risk of developing severe antivenom reactions. In such cases antivenom should not be given unless there are definite evidence of severe life threatening systemic envenoming. Reactions may be lessened by pretreatment with adrenaline, antihistaminics and corticosteroids. Rapid desensitization is not advised[19].

INTTIAL DOSE OF SOME IMPORTANT ANTIVENOMS [23]

Sl.No	Latin name	English name	Antivenom	App initial dose
1	Acanthophis species Bitis arietans	Death adder Puff adder	Monospecific Poly specific	3000-6000units 80 ml
2	Bothropsjararaca	Jararaca	Poly specific	20 ml
3	Bungarus caeruleus Calloselasma(Agkistrodon) Rhodostoma	Common krait Malayan pit Viper	Polyspecific Monospecific	100 ml 100 ml
4	Crotalus adamanteus	Eastern diamondback rattlesnakes	Polyspecific	30- 100 ml
5	Crotalus atrox	Western diamondback rattlesnakes	Polyspecific	30-100 ml
6	Crotalus viridis subspecies	Western rattle snakes		30-100 ml
7	Daboia (vipera) russelii	Russell's vipers	Monospecific Polyspecific	40 ml 100 ml
8	Echis species	Saw-scaled or carpet vipers	Monospecific Polyspecific	20 ml 100 ml
9	Hydrophilidae	Sea snakes	Monospecific	1000 units
10	Naja kaouthia	Monocellate Thai cobra	Monospecific	100 ml
11	Najanaja	Indian cobra	Polyspecific	3000-6000units
12	Notechis scutatus	Tiger snake	Monospecific	3000-6000units
13	Oseydecgus textiles	Eastern brown snake	Monospecific	12000units
14	Oxyuranus scutellatus	Taipan	Monospecific	100 ml
15	Trimeresurus albolabris	Gree pit viper	Monospecific	10 ml
16	Vipera berus	European adder	Polyspecific	50 - 80 ml
17	Vipera palaestinae	Palestine viper	Monospecific	

MORTALITY AND MORBIDITY

Untreated snake bite mortality is hard to assess, . Deaths are most unsual within a hr of the bite [10]. Late deaths more than 5 days after the bite usually result from renal failure**.** Death after snakebite may occur as rapidly as few minutes or as long as 41 days (King cobra and E.carinatus respectively). Most elapid deaths occur several hours after the bite. Most sea snake bites cause death between 12 — 14 hr, and viper bites within days.

The functional disability following pit viper bite about 32%. Death following pit viper venom poisoning is most likely to occur at 6 - 48 hr. following envenomation. The death rate is about 64%. Less than 17% of deaths occur within 6 hr, and less than 4% within 1 hr[<38]. Prognosis appears worst in infants and in elderly[19].

There has been significant reduction in mortality since the introduction of antivenin. Prior to the availability of the antivenin the mortality rates ranged from 5 - 25% [38]. The mortality rate for patients treated with antivenin is approximately 0.28%, compared to 2.6% for patients who do not receive antivenin, a statistically a significant difference[3] . The reasons for poor outcome in poisonous snake poisoning are delayed presentation, inadequate fluid resuscitation, inappropriate use of vasopressors, delayed or inadequate use of antivenin[38].

The average natural mortality following elapid bites is 10%, following viper bites is 1-15% and 10% for sea snake bite[42].

CAUSES OF DEATH IN SNAKE BITE :[23]

1. Respiratory arrest.
2. Sudden cardiac arrest
3. hypotension and shock.
4. Severe bleeding including intracranial bleeding
5. Acute renal failure
6. Septicemia

MATERIALS AND METHODS

The cases for present study were taken from the Department of General Medicine, R.L. Jalappa .Hospital,. The cases were studied from DEC 01 2008 onwards till NOV 30 2009 of snake bite and its hematological and Neurological Complications were completed. 79 snakebites were carefully examined and monitored for hematological and Neurological complications, and were encounted.

SOURCE OF DATA:

All cases were taken from Wards of Department of Medicine,R. L. Jalappa. Hospital, Tamaka, Kolar, the cases were closely monitored and vigilized from the day of admission to the day of discharge.

METHOD OF COLLECTION OF THE DATA:

It was Hospital case series study in which 50 patients who gave history of snake bite or had signs of envenomation or fang marks were selected. Out of 79 cases 50 cases who met the above criteria were taken from a period of 1 dec2008 - Nov 30 2009

INCLUSION CRITERIA:

1. Patients above the age of 18.
2. Patients presenting with history of snake bite.
3. Presence of fang marks.
4. Development of local or systemic reactions.

EXCLUSION CRITERIA:

1. Patient below the age of 18 years.

Sampling

The cases of snake bite with hematological or neurological complications were taken and analysed from 1 dec2008 til 30 nov 2009.

INVESTIGATIONS AND INTERVENTIONS:

1) Blood examinations for various investigations of hematological investigations.

2) Chest x-ray was taken in all the patients.

3) ECG was taken in all patients.

4) It was decide to do Lumbar Puncture if the patient developed neurological signs of intracranial hemorrhage. In the present study there was no need for Lumbar Puncture.

5) Anti snake venom was given to the patients with signs of local or systemic envenomations. ASV given in the present study is manufactured by Central Research Institute, Kasauli and is polyvalent antivenin. This drug is approved for the use in India. Inspite of known complications of the antivenin, the benefits over score the adverse reactions.

Detailed history was taken and clinical examination was done in all patients and records on a detailed proforma approved by the Guide. Regular follow-up was done during the hospital stay, and after discharge.

Attempt was made to identify the snakes based on the description given by the patient, showing the photographs of the poisonous snakes and by fang marks.

Antibiotic coverage chiefly for anaerobic organisms was done in all cases whether or not patient developed fever and signs of systemic infection. All patients were put on analgesics and anti-inflammatory drug.

Surgical opinion and intervention was sought whenever needed.The results were analyzed and compared with other studies (both Indian and Western) and discussed.

The hematological investigations included the following:

1) Hb%
2) Total count
3) Differential count
4) Bleeding time (BT)- every day till the values returned to normal.
5) Prothrombin time (PT) - every 2" day till the values returned to normal
6) aPTT - every 2 "d day till the values returned to normal.
7) Platelet count
8) Urine microscopy
9) Stool routine

Other investigations included the following:

1. blood urea
2. serum creatinine
3. Chest x-ray
4. ECG
5. CT Scan brain.

All patients of snakebites were admitted and kept under observation for the development of features of envenomation. 0.5 ml of Tetanus Toxoid was given to all the patients.

In known poisonous snake bites and in patients developing features of envenomation 5 to 10 vials of polyvalent antivenom was administered in IV infusion without any skin test to avoid any delay in the treatment. In patients developing reactions, antihistaminics and steroids were given in appropriate doses. Adrenaline was not used in any of the cases, as the reactions were mild. Depending upon the severity of the poisoning and course of the illness further ASV was given

RESULTS AND ANALYSIS

NUMBER OF CASES OF HEMATOLOGICAL AND NEUROLOGICAL COMPLICATIONS IN SNAKE BITE:

The cases were taken from 1 December 08 to 30 NOV 09.

	No. of cases	PERCENTAGE
Hematological complications	13	26
Neurological complications	13	26
Local Envenomation	17	34
No envenomation	7	14
Total	50	100

The total number of snake bite dmissions in the medical wards during that period was 79 out of which 50 cases were taken up for the study out of which 26% had hematological and neurological complications. Local envenomation was seen in 34%. No envenomation in 14%.

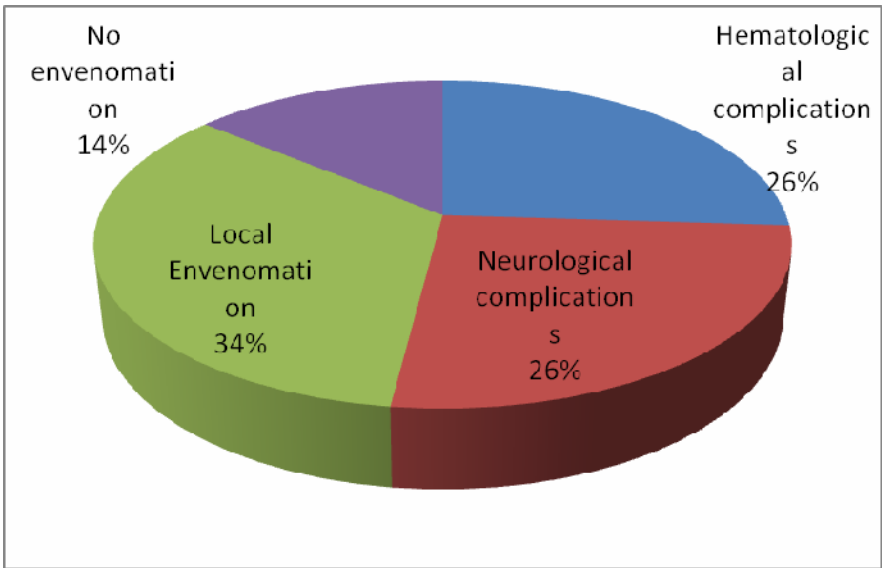


TABLE : 1

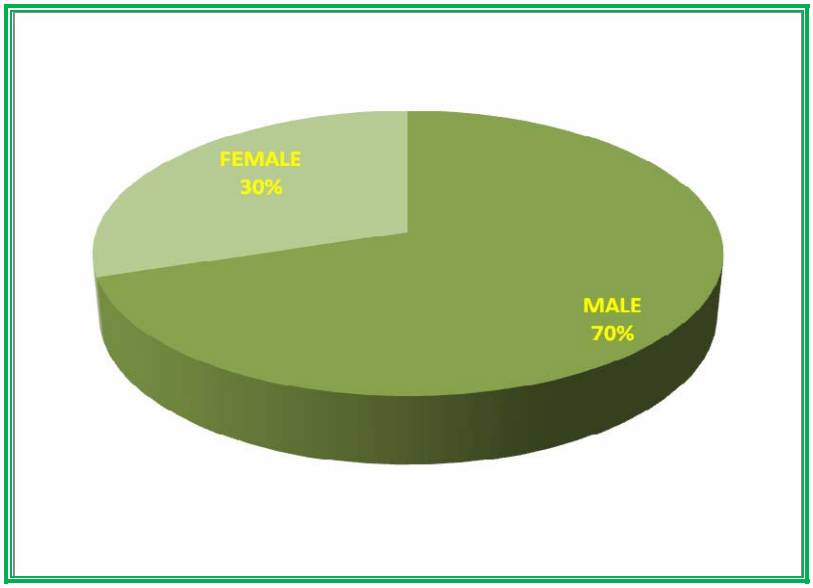
INCIDENCE OF SNAKE BITE ACCORDING TO SEX

AGE GROUP (in years)	NO. NO OF CASES	PERCENTAGE
18-19	1	2%
20-29	13	26%
30-39	13	26%
40-49	8	16%
>50	15	30%

INCIDENCE OF SNAKE BITE ACCORDING TO SEX

SEX	NO. OF CASES	%
MALE	35	70
FEMALE	15	30

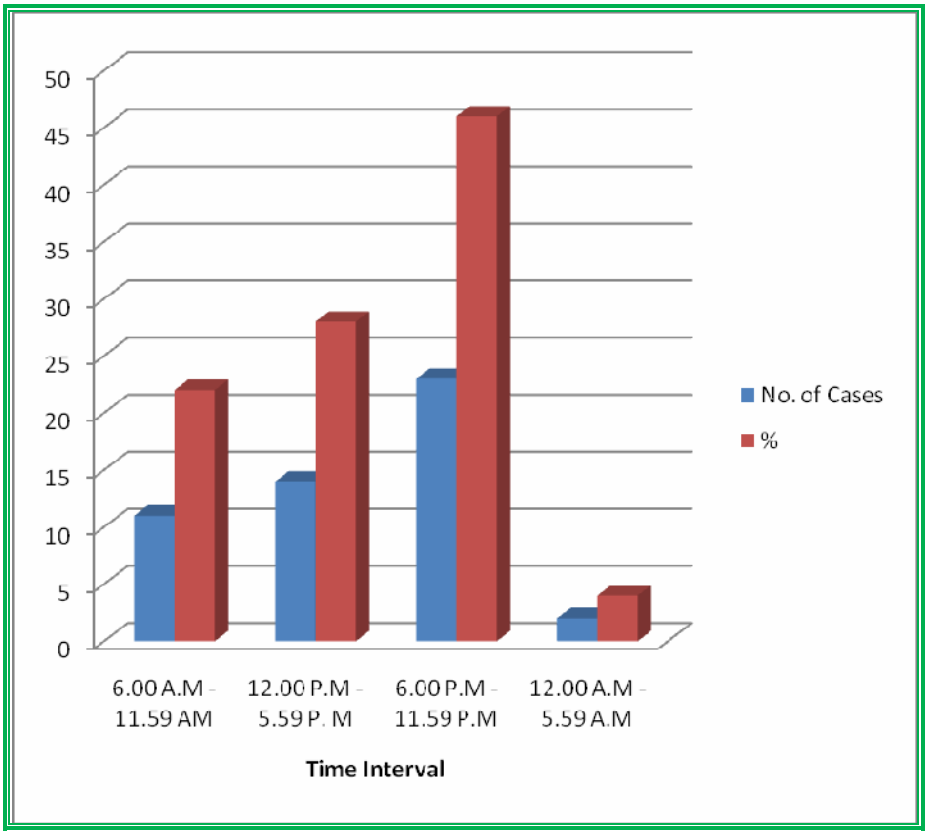
Male patients out numbered the female patients



APPROXIMATE TIME OF SNAKE BITE

TIME INTERVAL	No. of Cases	%
6.00 A.M - 11.59 AM	11	22
12.00 P.M - 5.59 P. M	14	28
6.00 P.M - 11.59 P.M	23	46
12.00 A.M - 5.59 A.M	2	4

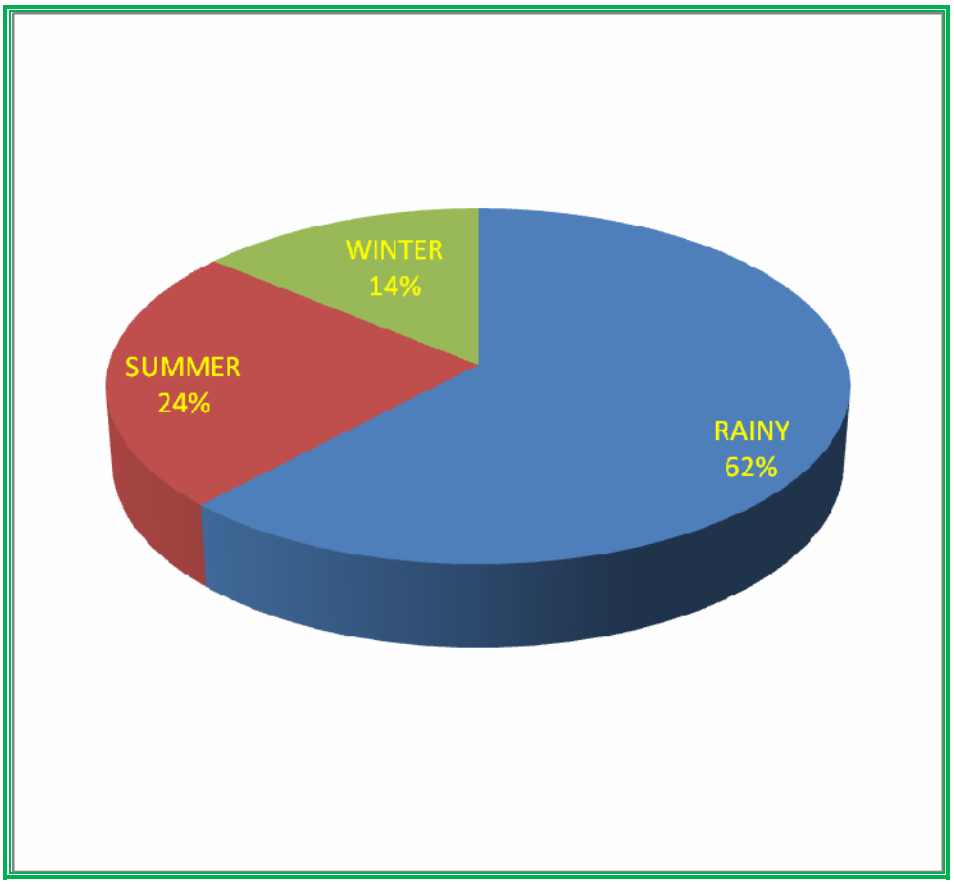
In this study 46% of the cases were seen between 6pm-12am that is due to poor visibility in the dark.



SEASONAL TRENDS OF SNAKE BITE:

SEASON	NO. OF CASES	%
RAINY	31	62
SUMMER	12	24
WINTER	7	14

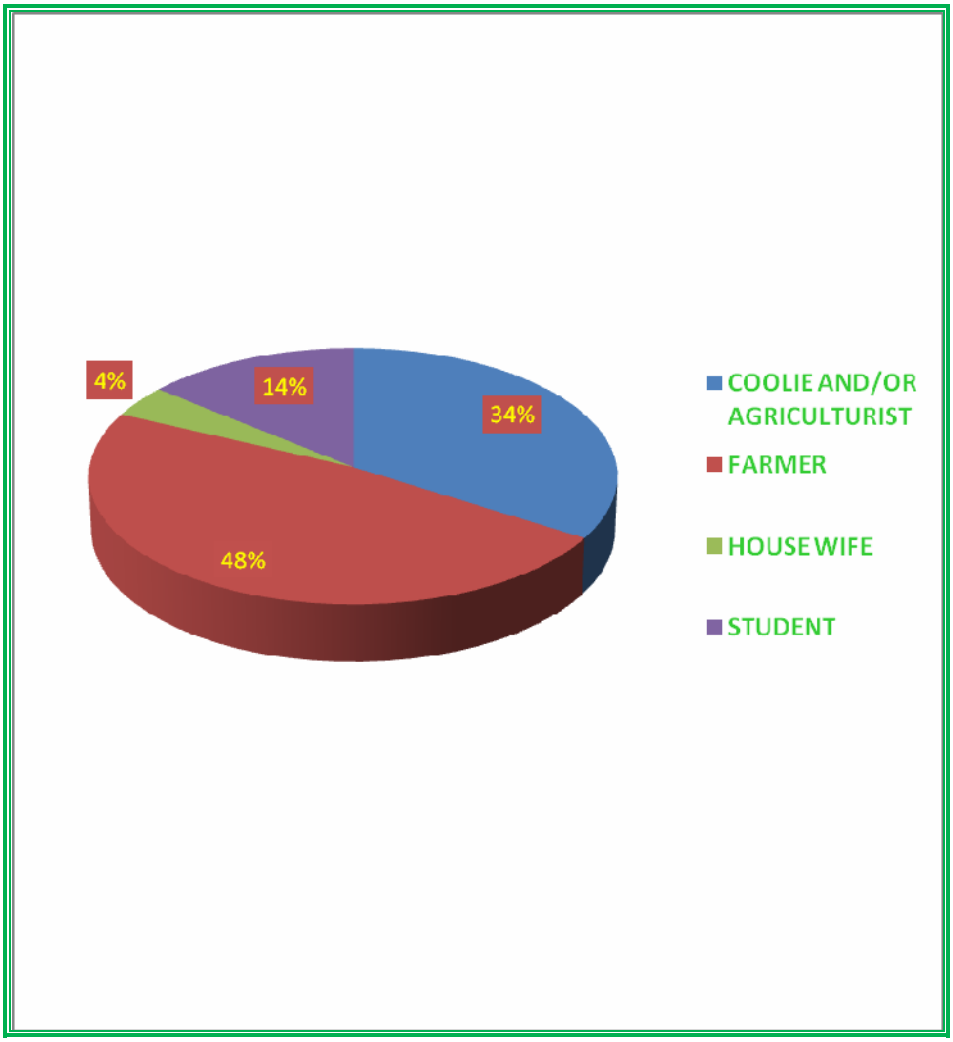
In this study 62% of the cases were in rainy season and only 14% in winter.



OCCUPATION OF PATIENTS

OCCUPATION	NO. OF CASES	%
COOLIE AND/OR AGRICULTURIST	17	34
FARMER	24	48
HOUSE WIFE	2	4
STUDENT	7	14

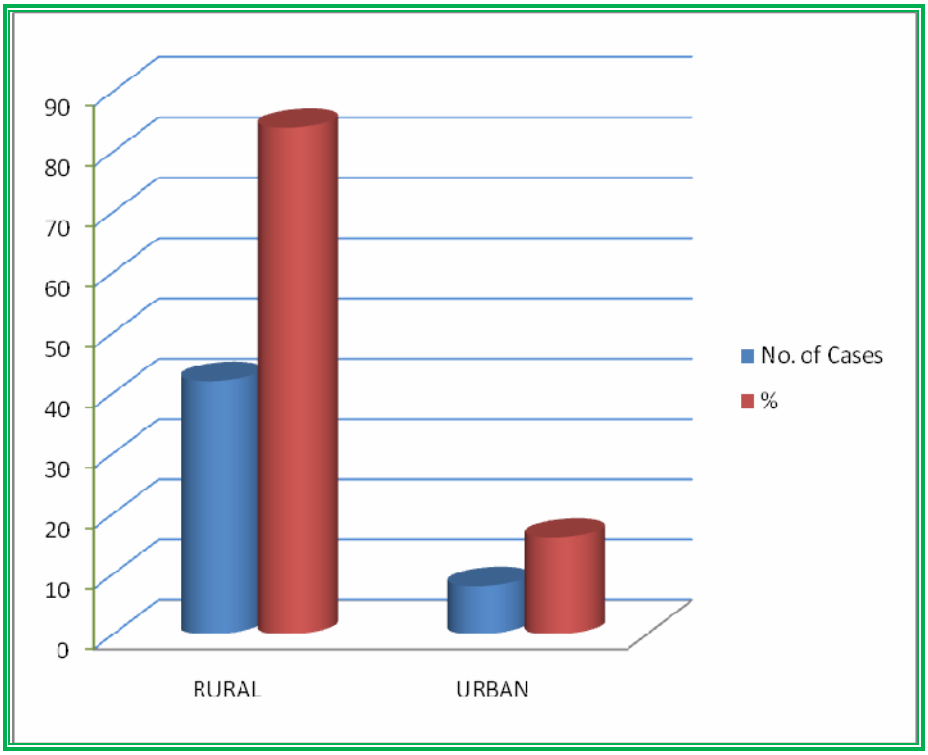
In this study most of the snake bites were seen in the people who work in the fields, including farmers, coolie workers and agriculturists.



RURAL URBAN INCIDENCE

	NO. OF CASES	%
RURAL	42	84
URBAN	8	16

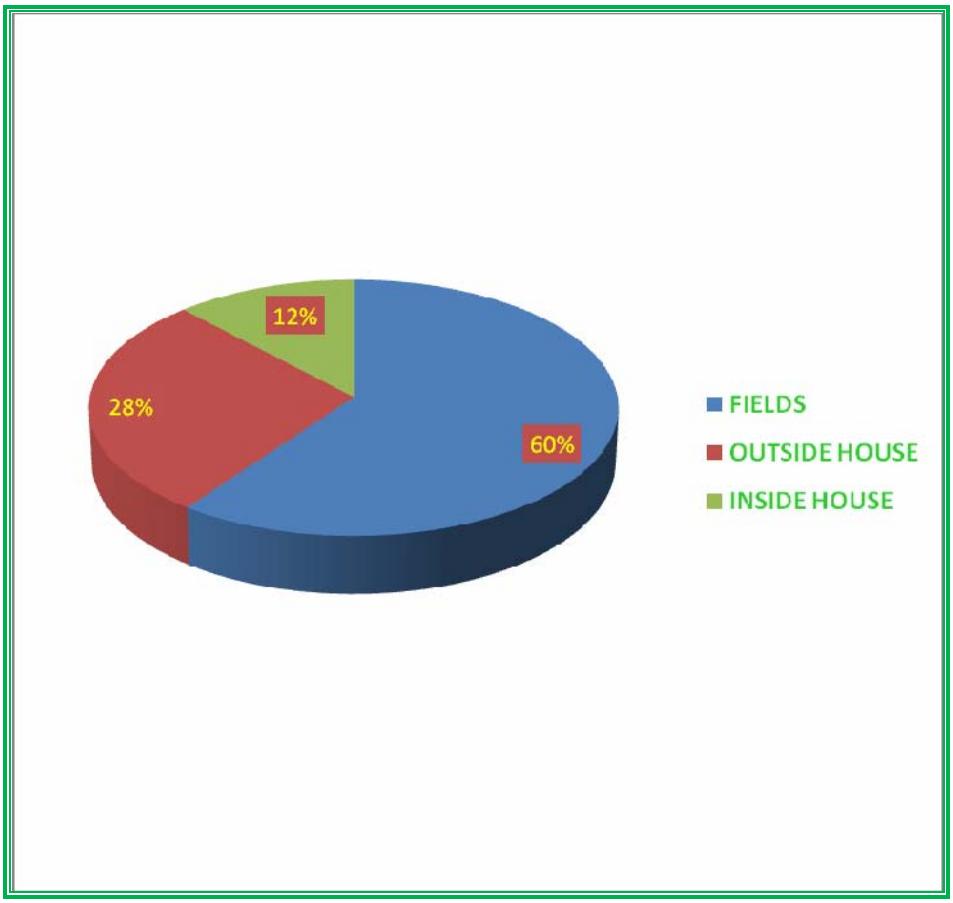
In this study 84% of the cases were from the surrounding villages of the kolar and semiurban areas. Only 16% were from Kolar proper.



PLACE OF SNAKE BITE

PLACE	NO. OF CASES	%
FIELDS	30	60
OUTSIDE HOUSE	14	28
INSIDE HOUSE	6	12

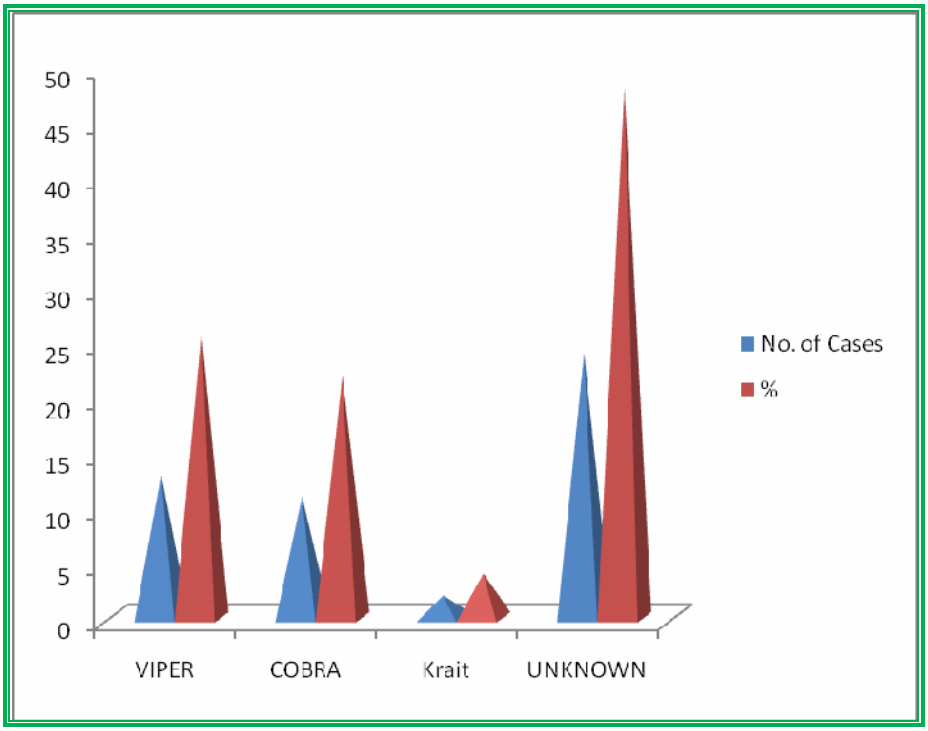
88% of the snakebites occurred outside the house, out of which nearly 60% of the bites occurred in the fields. Only 12% of the snakebites occurred in the house.



TYPES OF SNAKE

TYPE	NO. OF CASES	%
VIPER	13	26
COBRA	11	22
Krait	2	4
UNKNOWN	24	48

Six snakes were brought to the hospital. The snakes were identified based on the patients description and identification of the snakes. 26% of the snake bites were identified as viper bites where as 22% were identified as cobra bite. Rest of the unidentified snakebites can be presumed as viper bites and cobra because of hematological and Neurological manifestations.



FIRST AID RECEIVED BY THE PATIENTS

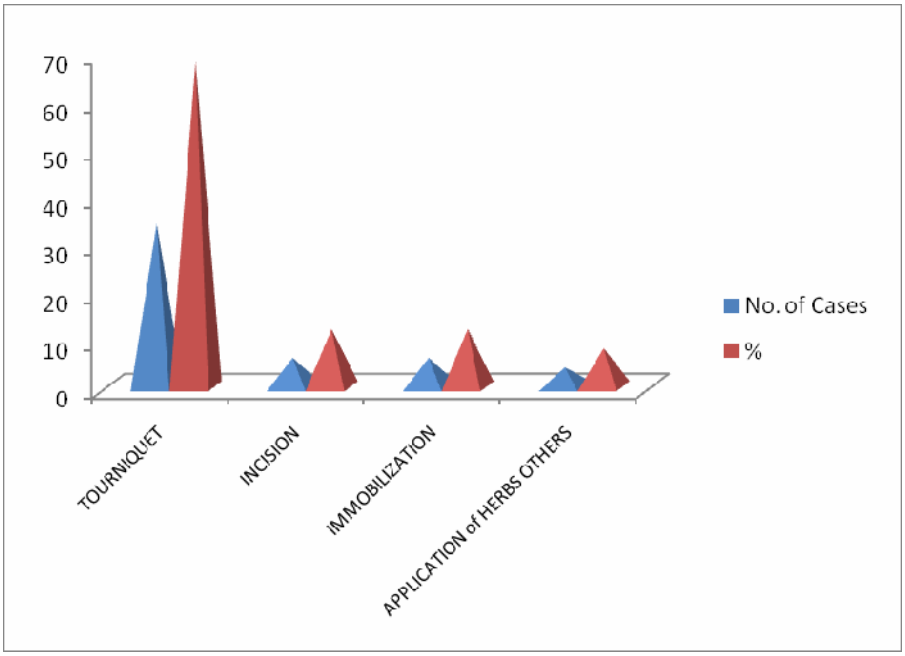
FIRST AID	NO. OF CASES	%
RECEIVED	46	92
NOT RECEIVED	4	8

92% of the patients received First Aid, which suggests the awareness of snakebite complications.

FIRST AID METHODS

FIRST AID	NO. OF CASES	%
TOURNIQUET	34	68
INCISION	6	12
IMMOBILIZATION	6	12
APPLICATION of HERBS OTHERS	4	8

Application of the tourniquet was the most common first aid measure (68%). Incision at the site of bite was done in 12% of the cases. Awareness about immobilization of the bitten part was less, accounting for only about 12% of the cases, out of 4 cases of application over the site of the bite, most of the cases applied herbs. Few cases applied dung of cows/ buffaloes.



TIME INTERVAL BETWEEN SNAKE BITE AND ADMISSION

TIME IN HOURS	NO. OF CASES	%
0-2	10	20
2.1-4	16	32
4.1-6	9	18
6.1-8	4	8
8.1-10	2	4
10.1-12	1	2
12.1-24	6	12
>24	2	4

Most of the cases were admitted within 2 -4 hours of the bite accounting for 32% of the cases. 84% of the patients were admitted within 12 hours whereas 96% were admitted within 24 hours. Only 4% of the patients were admitted after one day.

SITE OF BITE

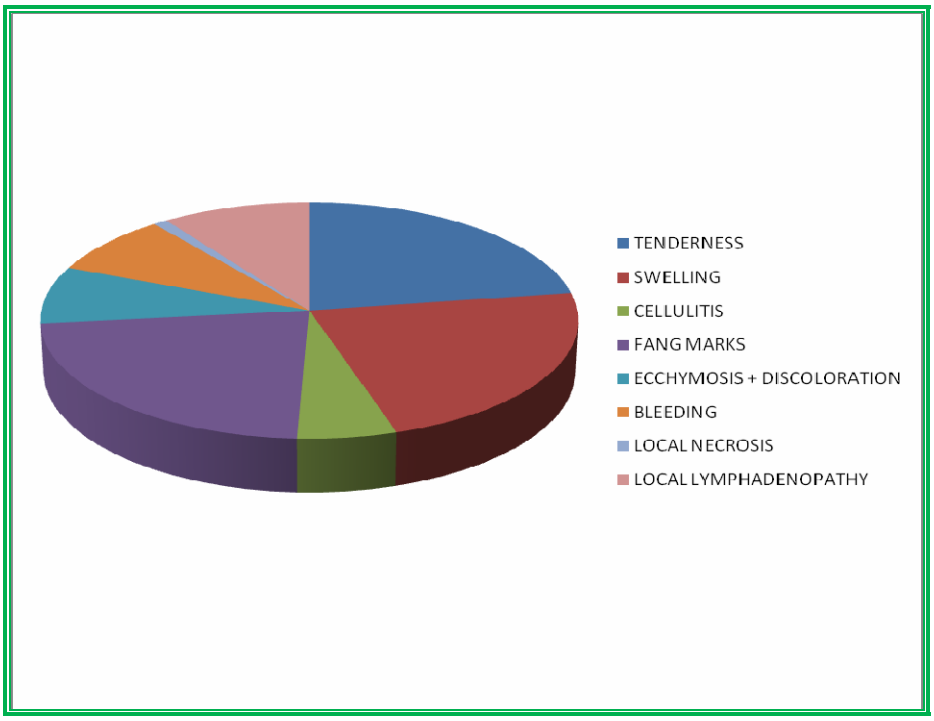
SITE OF BITE	NO. OF CASES	%
Left foot	26	52
Left hand	2	4
Right foot	17	34
Right hand	2	4
Right shoulder	1	2
Right buttock	1	2
Right thigh	1	2

90% of the snake bites were in the lower limbs in that 86% were in the foot.

LOCAL EXAMINATION

LOCAL EXAMINATION	NO. OF CASES	%
TENDERNESS	49	98
SWELLING	49	98
CELLULITIS	12	24
FANG MARKS	49	98
ECCHYMOSIS + DISCOLORATION	17	34
BLEEDING	18	36
LOCAL NECROSIS	2	4
LOCAL LYMPHADENOPATHY	21	42

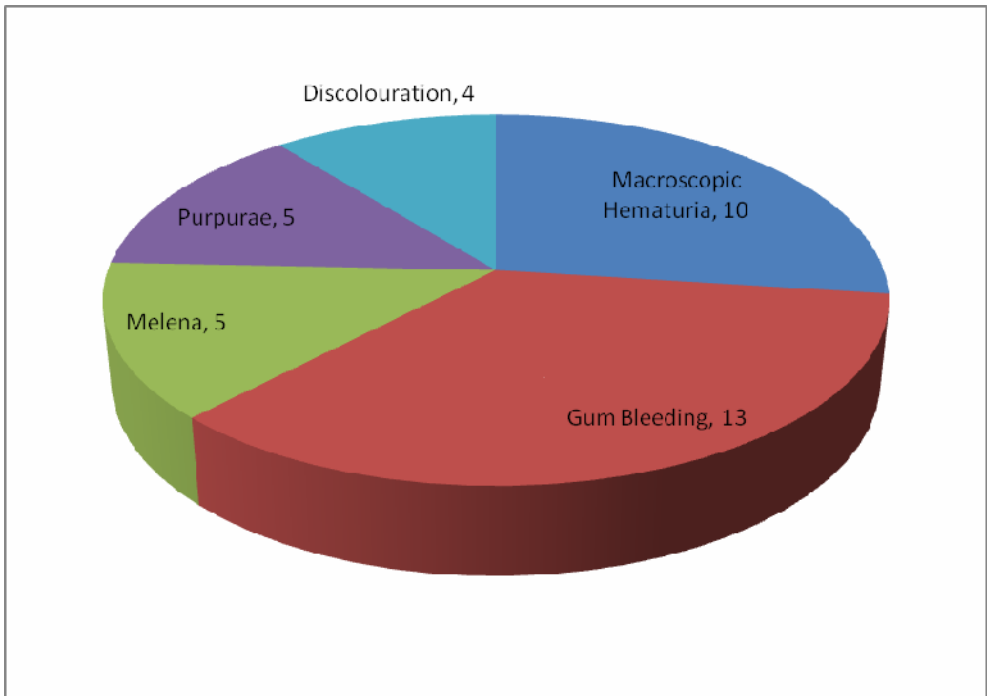
Tenderness and local swelling were found in almost all cases. Bleeding from the site of bite during examination was found in about 36% of the cases. Fang marks were seen in 98% of-the cases. Local necrosis at the time of examination was seen in 4% of the cases. Lymphadenopathy was seen in 42%.



OUT OF 13 NEUROLOGICAL CASES,THE MANIFESTATIONS WERE

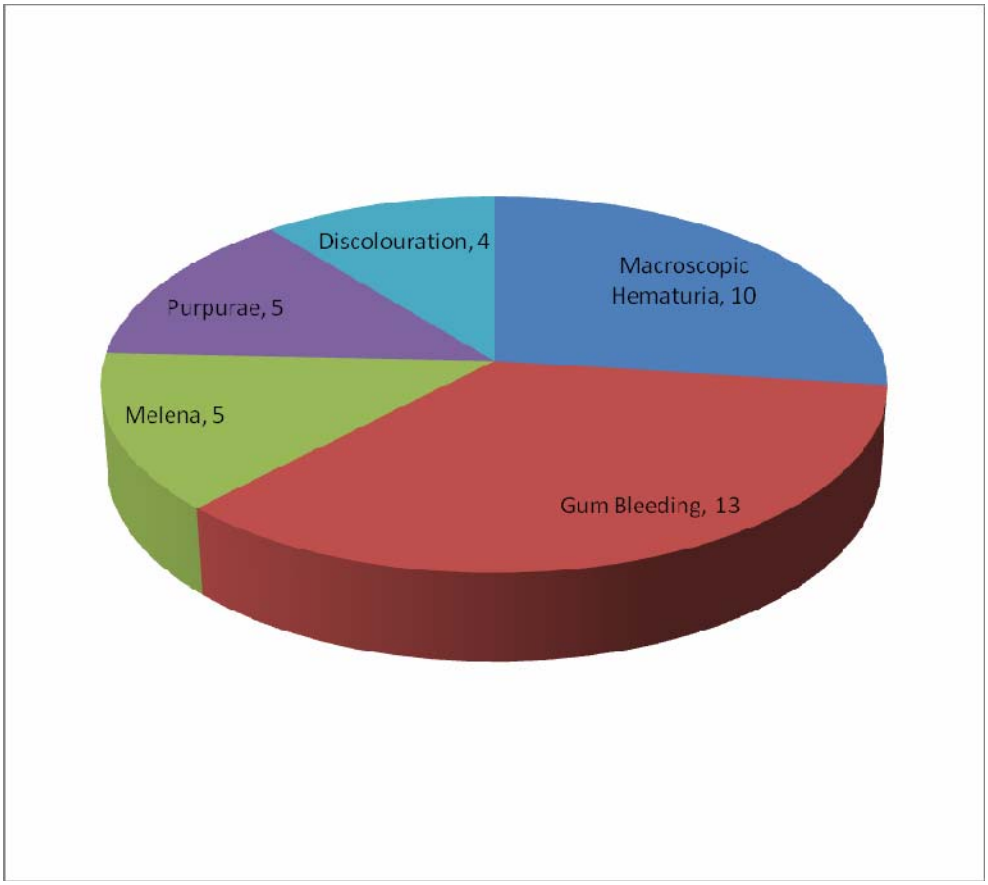
Neurological manifestations	No. of cases	%
PTOSIS	13	100
DIPLOPIA	13	100
DIFF IN SWALLOWING	6	46
GIDDINESS	1	8
MOTOR DEFICITS	1	8

All the patients in this study had bilateral ptosis and diplopia. 50% of the patients had difficulty in swallowing. 1 patient had left sided hemiplegia.



OUT OF 13 HEMATOLOGICAL CASES, THE MANIFESTATIONS WERE

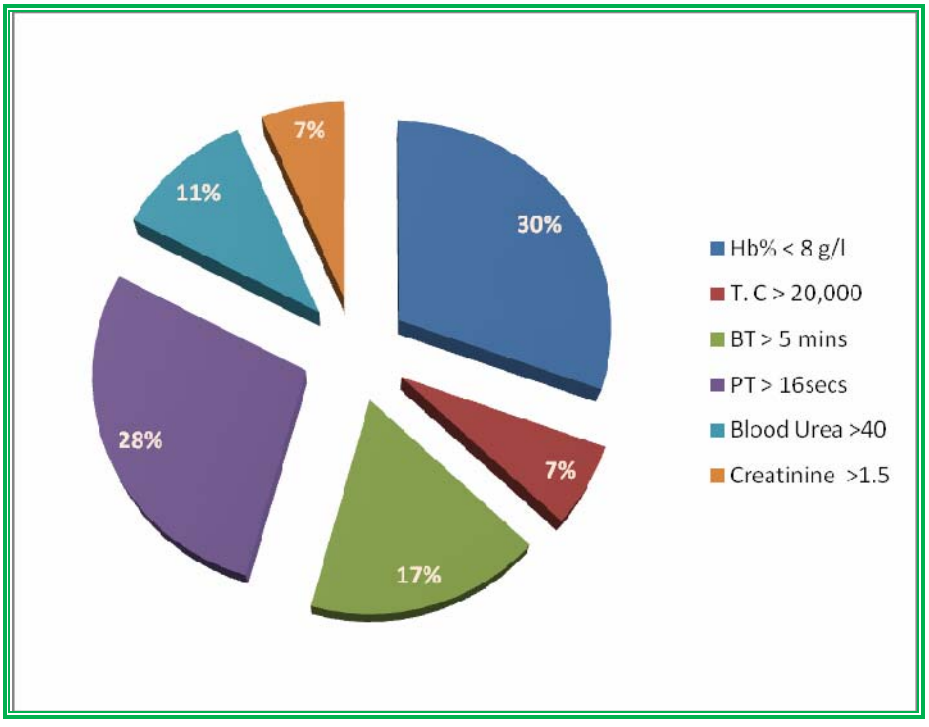
HEMATOLOGICAL MANIFESTATIONS	NO OF CASES	%
Macroscopic Hematuria	10	77
Gum Bleeding	13	100
Melena	5	38
Purpurae	5	38
Discolouration	4	31



INVESTIGATIONS

Investigations	No of cases	%
Hb% < 8 g/l	14	28
T. C > 20,000	3	6
BT > 5 mins	8	16
PT > 16secs	13	26
Blood Urea >40	5	10
Creatinine >1.5	3	6

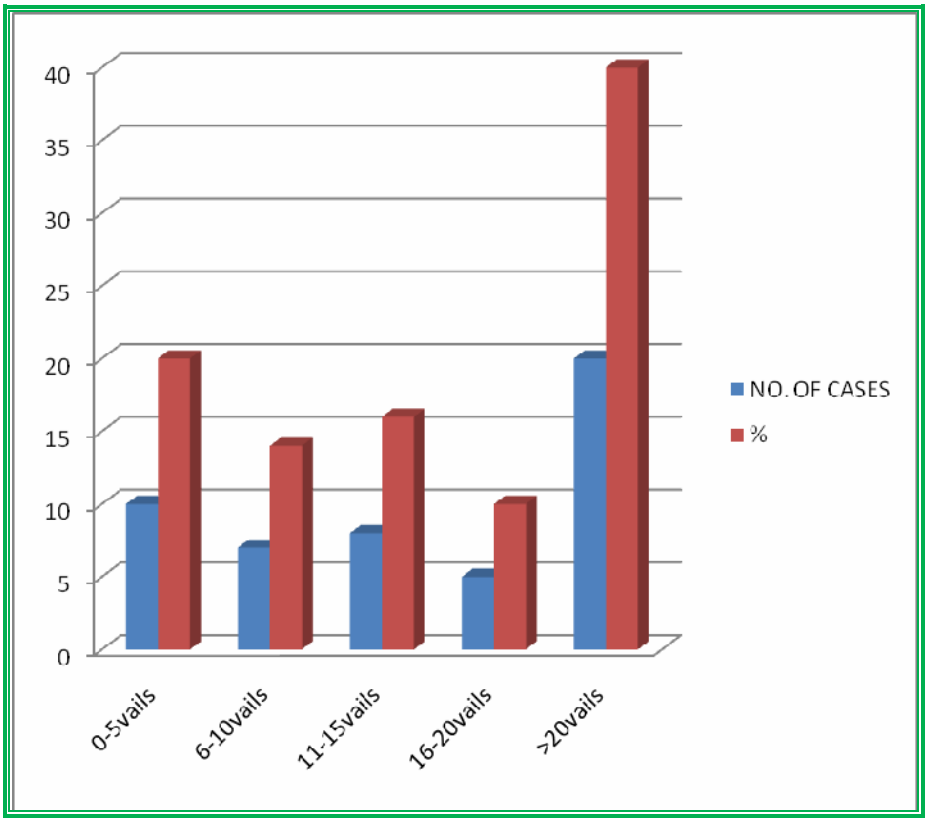
In all the cases of snake bite hemoglobin was less in 28% of cases, Total count >20,000 was seen in only 6%, Prothrombin time is elevated in 26%,and Acute renal failure in only 10%.



NUMBER OF ANTI-SNAKE VENOM GIVEN

NO of VAILS	No. of cases	%
0-5vails	10	20
6-10vails	7	14
11-15vails	8	16
16-20vails	5	10
>20vails	20	40

In this study 40% of the cases were given more than 20 vails of ASV.



DURATION OF HOSPITAL STAY

DURATION	No. of cases	%
1-5 days	29	58
6-10days	17	34
11-15days	1	2
AMA	2	4
DEATH	1	2

Half of the patient stayed between 1-5 days. Two – third of the patients were discharged within 10days.2 were discharged against medical advice and 1 mortality was seen in this study.

DISCUSSION

In the present study 26% of the poisonous snakebite cases had hematological complication and 26% had neurological complications. 2 cases, which had hematological complications, had neurological complications.2 patients had local, hematological, neurological , ARF, and local cellulitis and had to under go fasiotomy.

AGE INCIDENCE

AGE GROUP (in years)	PRESENT STUDY	REID ET AL(45;)	BANERJEE ET AL
18-19	12%	23%	19.56%
20-29	20%	9.6%	48.55%
30-39	24%	11.5%	11.41%
40-49	14%	28.8%	5.7%
50-59	14%	15.3%	1.42%
60-69	10%	11.5%	5.7%
>70	6%	NIL	NIL

The cases ranged from 18 years to maximum of 75 years with mean of 46 years. The present study shows that about half of patients are within the age group of 18-40 years. Other studies show more common age group of <30 years.

SEX INCIDENCE

SEX	PPESENT	RE ID ET AL (45)	SAINI ET AL (50)
MALE	70%	72% -	73.3%
FEMALE	30%	28%	26.6%

SEX RATIO

PRESENT STUDY	MITRAK UL ET AL(32)
2.4 :1	14 :1

Males are the victims for snakebite more often than females in all the studies including the present study. The percentage of males ranged from 54.8% to 75% in various studies and present study shows a male preponderance with 70% which is in close relation with other studies. The male to female ratio in present study is 2.4 : 1.

TIME OF BITE

TIME INTERVAL	PRESENT STUDY	MITRAKUL ET AL
12-A-M -5.59 A.M	4%	NIL
6 A.M- 11.59 A.M	22%	70%
12 P.M.-5.59 P.M	28%	10%
6 P.M - 11.59 P.M	46%	20%

Majority of the snake bites occurred between 12 P.M to 11:59 P.M accounting for 74% of the cases compared to only 30% in Mitrakul et al. Only 4% snake bite cases were in morning hours while Mitrakul et al had no cases at that time. This suggests that snake bites are common during working hours of humans unlike the nocturnal hours of snake.

PLACE-OF BITE

PLACE	PRESENT STUDY	MITRAKUL ET 1 AL(38)
"FIELDS	88%	80%
HOUSE YARD	12%	10%
NO RECORD	NIL	10%

Majority of the snakebites occurred in agricultural fields in the present study and also in the study done by Mitrakul et al. 12% of the snakebites occurred inside the house in the present study due to unhygenic houses and pets within the house.

SNAKE IDENTIFIED

PRESENT STUDY	SAJNI ET AL(25)
44%	55%

The majority of the snakebites in the present study was by cobra and viper. Six snakes was brought to the hospital. Based on the patient's description, 44% of the snakes were identified and majority of them were cobra. In the study done by Saini et al, snakes were identified in *55%* of the cases.

INTERVAL BETWEEN BITE AND ADMISSION

INTERVAL (IN HRS)	PRESENT STUDY	MITPKUL ET AL(38)
30MIN - 3 HOUR	22%	40%
3-HOUR - 8 HOUR	56%	60%

About 78% of the cases were admitted between 30mins to 8 hours in present study and in Mitrakul et al study it was 100%.

SEASONAL TRENDS OF SNAKE BITE

SEASON	PRESENT STUDY	VIRMANI ET AL(63)
RAINY	62%	56.6%
SUMMER	24%	13.2%
WINTER	14%	30.2%

In present study majority of snake bites were in rainy season which correlates with virmani et al study, next common was in summer season compared to winter in virmani et al study.

RURAL/URBAN INCIDENCE

	PRESENT STUDY	VIRMANI ET AL(63)
RURAL	84%	93.9%
URBAN	16%	6.1%

Most of the snake bites occurred in rural areas accounting for 88.6% to93.6% in various studies, the present study showed 84%.The rural to urban ratio is 5.25:1.The high incidence of snake bites in rural is due to unhygienic houses, fields near by, pets close to the houses and agriculture as there main occupation.

SITE OF BITE

SITE OF BITE	PRESENT STUDY	MITRAKUL ET AL(38)
LOWER LIMB DISTAL	86%	80%
UPPER LIMB DISTAL	4%	10%
LOWER LIMB PROXIMAL	4%	NIL
UPPER LIMB PROXIMAL	2%	10%
OTHERS	NIL	NIL

Most of the snakebite were observed in distal part of the limbs in all the above mentioned studies including the present study. Distal part was bitten in 90% in the present study whereas in other studies it ranged from 82% to 97% . The distal part of the lower limb is bitten in 86% of our study where as in other studies it ranged from 21% to 80%.

LOCAL SYMPTOMS

SIGNS	PRESENT STUDY	MITRAKUL ET AL(38)	VIRMANI ET AL(39)
PAIN	98%	100%	63%
SWELLING	98%	100%	63%
FANG MARKS	98%	70%	80%
LOCAL -NECROSIS	8%	10%	1.3%
-BLISTER	6%	10%	27%

Pain and swelling were the most common symptoms in the present as well as the other studies, accounting for nearly 100%. Fang marks were noticed in 30% to 73% of cases in various studies, but in the present study fang marks were noticed in 98% of the cases. Local necrosis was found in 0-11% in various studies and in present study it is 8%. Blisters are found in 6% of cases in present study and it ranges from 13-42% in the other studies mentioned above.

Hematuria and gum bleeding was the most common presentation among hematological manifestation in the present study. Similar results were seen in other studies like that of Saini, Sarangi and Bhat Hematuria was observed in 73 to 88.67% of the cases in various studies. Most of the Indian studies have shown hematuria as the most common manifestation.

Ecchymosis and discoloration were found in 40 to 100% of the cases as mentioned in above studies and in the present study, the values fall within the above mentioned range (40%).

Bleeding from other than the site of bite and gum bleeding was not a common observation in the present study where as it is a common observation in studies of Reid et al and Mitrakul et al. In Indian studies, bleeding from other than the site of bite was not a common observation as seen in Saini et al and Sarangi et al. Melena was seen in 40% in present study.

1 case in present study showed clinical evidence of Subarchnoid hemorrhage so CT Scan brain was done and that was the only patient died accounting for 2% mortality. In the other studies, SAH ranged from 0 to 4. 1 % of the cases, accounting for 2- 4% mortality.

In present study none of the cases manifested with the features of DIC. In saini et al 20% of cases had DIC.

VOMITING

PRESENT STUDY	SARANGI ET AL	WARELL ET A1 (40)	MITRAKUL ET AL(38.)
22%	88.6%	28%	30%

Vomiting was seen from 28 to 88.6% of the cases in various studies as compared to 22% in our study.

LYMPHADENOPATHY

PRESENT STUDY	WARELL ET A1	BHAT RT AL(14)	SARANGI ET AL
42%	88.6%	28%	30%

Local lymphadenopathy was seen in 42% of the present study, where as in other studies it ranged from 27 to 77% of the cases.

HYPOTENSION

PRESENT STUDY	WARELL ET AL(40)
NIL	4%

In our study none of the patient, suffered from hypotension. In Warrel et at study 4% of the cases had hypotension.

Hb%(ANEMIA)

Hb%	PRESENT STUDY	SAINI ET AL(25)	SARANGI(41)
<5	Nil	20%	NIL
5-10	32%	20%	16.6%

One third of the patients had anemia. But it is difficult to assess the anemia secondary to bleeding disorder.

TC

	PRESENT STUDY	REID ET AL(35)	SAINI ET AL(25.
NORMAL	60%	100%	60%
LEUCOCYTOSIS	38%	NIL	40%
LEUCOPENIA	2%	NIL	NIL

Most of the cases showed normal total WBC count accounting for 60% of the cases. In study of Reid et al all cases had normal WBC count where as in Saini et al leukocytosis was found in 40% of the cases. In present study leukocytosis was found in 38%'of the cases.

BT:> 4 MINs

PRESENT STUDY	SAINI ET AL(25)
36.3%	16.6%

Bleeding time was taken as abnormal if it is more than 4 minutes and was abnormal in 36.3% of the cases in the present study. In the study of Saini et al it was 16.6%.

PT: >14 SEC

PRESENT STUDY	SAINI ET AL(25)
38%	100%

PT was abnormal in 38% of the cases.

CHEST X-RAY

PRESENT STUDY	REID ET AL (35)
NORMAL IN ALL	NORMAL IN ALL

Chest X-ray was normal in all cases in present as well as Reid et al study.

MORTALITY

PRESENT STUDY	MITRAKUL ET AL (38)	BHAT ET AL	WARR EL ET AL	NIGAM ET AL	SARANGI ET AL
2%	NIL	1.96%	4.3%	8.3%	8.3%

In present study 2% mortality was found. In sarangi et al mortality was 8% and in Mitrakul et al there was no mortality seen.

CONCLUSIONS

A Hospital case series study of 50 cases of poisonous snakebites with hematological and Neurological complications were selected among 79 snakebites cases admitted in the wards of Medicine, R. L. Jalappa Hospital from 1 december 08- november 31 2009. A detailed history, a methodical clinical profile, established treatment and follow-up was done in each case and recorded.

There was definite male preponderance and most of the patients were agricultural workers with rural background of adult age group. The snakebite was common in rainy season. Most of the hematological complications were secondary to viper bites and occurred while working in the fields during daytime.

Most of the patients received first aid within 30 minutes of the snakebite, which is an important observation in the present study. This shows an increased awareness of the first aid in the management of the snakebites in rural population also. Tourniquet was tied in most of the cases, while incision and applying herbs was carried out in many. Very few were aware of the importance of immobilization of the bitten part. Most of the patients were admitted within the 8 hours of the bite.

Pain and the swelling were the most common clinical manifestations followed by bleeding from the site of the bite. The hematological complications other than bleeding from the site of the bite (systemic) were hematemesis, melena,gum bleeding and macroscopic hematuria. However none of the cases showed severe and prolonged hematological abnormality. In present study there were no cases of hypovolemic shock, or DIC. Neurological complication that were seen are ptosis, dysphagia, diplopia and 1 patient had left sided hemiplegia due to sub arachanoid hemorrhage.

The systemic complications other than hematological were solely related to renal system. The renal complications were likely to be secondary to the direct venom toxicity as none of the cases had hypovolemia.

Gum bleeding and Macroscopic hematuria were the most common hematological manifestation of the poisonous snakebite (26% of the cases) Ptosis and diplopia were the most common neurological manifestation. Bleeding time, prothrombin time were carried out in all the patients. Other investigations like Hb%, total count, differential count, platelet count, routine urine examination, ECG and screening of the chest were also carried out in all the patients. BT, PT and PTT were carried out during the course of the treatment.

All patients were given antivenins according to the grading and progression of symptoms and signs. Mild early reactions were noticed in few cases, but in none of the cases there was severe reactions. In this study 40% of the cases were given more than 20vails of ASV. Supportive treatment like I.V fluids, tetanus toxoid, broad-spectrum antibiotic coverage and anti-inflammatory drugs were given in all patients. Two of the patients had Acute renal failure which gradually subsided with out dialysis. Fasciotomy was the most common surgical intervention. The hematological complications were monitored by serial BT, PT and the values returned to normal by 6 days in all of the cases.

There was one death in the present study. This can be explained by the fact that most of the patients received first aid within 30 minutes of snakebite and received the antivenin within 8 hours.

SUMMARY

The present study can be concluded with the following observations:

1. Since microscopic hematuria is the commonest hematological manifestation following poisonous snakebite, routine urine examination must be mandatory in all the snakebite cases to detect the hematological manifestations at the earliest.
2. Serial monitoring of BT, PT are enough to monitor the patient having hematological complications.
3. Antivenin in proper doses should be given in all cases of poisonous snakebites (better late than never .
4. No specific treatment other than antivenin is needed in hematological or neurological complications.
5. Ventilatory support along with Neostigmine may be need for the patient with Respiratory paralysis.

BIBLIOGRAPHY

1. Banerjee. R.N et al - Neostigmine in the treatment of elapidae bites. J.Asso. Phy. Indi.20: 1972.
2. Basu et al, Acute renal failure following snakebite (Viper), J.Asso.Phy. Ind. Vol : 25,Dec 1977
3. Warrell DA. Guidelines for clinical management of snake bites in South East Asian region. SEAMEOTROPMED - Regional Centre for Tropical Medicine, WHO; 1999. Available from: http://www.searo.who.int/LinkFiles/SDE_mgmt_snake-bite.pdf.
4. Bhat R.N Viperine snake bite poisoning in Jammu.Med.Asso. Dec 1974 ;63: 383 - 392.
5. Burger J.L.and Dart R.C., Snake venom coagulopathy use and abuse of blood products in the treatment of pit viper envenomation, Ann. Emer. Med. July 20 (7),745 -801 ; 1991 (Med)
6. Chaterjee. C.C, Human physiology,. Vol 1 , 14th ed, 1990 , Medical allied agency, Delhi, 131 - 32.
7. Chaterjee S.C., Management of Snake bite cases, J.In Med . Asso.,1965 , 45 ; 654 - 59.
8. S.C and Kunkel, D.B.Death from rattle snake bite. Am J. Emarg. Med. 1985 ; 300 : 27.
9. Snake-Bite-Protocol-2007-India.htm:delhi.
10. P.J, Snakes of India, 4th ed. (revised) 1990 , NBT, India.
11. Blackman,Venomous snakebite, past, present and future treatment options, J.Am. Board of FAM , 5(4); 399 - 1992 (MEO)

12. Edward.Et.al, Davidson's principles and practice of medicine, ELBS Churcill Levingstone pib, 17th ed., 44-45.

13. B.W,Emergency Surgery, 12th ed., Hamilton Bailey's ELBS Pub, 851 - 858.

14. M.J. et al , rapid detection of cross linked fibrin degradation products in plasma monoclonal antibody - coated latex particle, AJCP, Vol 85, 3 Mar 1989, 360-64.

15. Fauci, et al, Harrison's principles of internal medicine, 14th ed., Mc.Graw Hill Pub.

16. Krysa-Clark J, Lewis S, Waterworth TA. Management of a snake bite in the field. J R Army Med Corps 2004; 150:97-8.

17. Ganong, Human physiology, Appleton LangePub, 17th ed.

18. Bawaskar HS, Bawaskar PH, Punde DP, Inamdar MK, Dongare RB, Bhoite RR, et al. Profile of snakebite envenoming in rural Maharashtra, India. J Assoc Physicians India 2008;56:88-95.

19. Gordan Cook, Manson's tropical diseases, Saunder's publ., ELBS, 20' ed., 469 - 495.

20. Harvey et al, The principles and practice of medicine, 22" ed. 262.

21. Hati, A.K., Mandalh, De. M.K.et al. In epidemiolog of snake bite in the district of Burdwan, West Bengal, JIMA, 90 : June 1992 (145 - 147).

22. Hoffman. R et al, Hematology basic principles and practice, Churchill Leningstone Pubed., 73 3.

23. Jena Indramani and Sarangi Akulananda, Snakes of medical importance and snakebite treatment publication House. 1993.

24. J. Otten, Venomous animal injuries in emergency medicine — concepts and clinical practice Rosen and Barken, 3rd ed., 1992 byMobsy year book Inc.

25. Kelly, Text book of internal medicine, 2nd ed., 1578.

26. Brunda G, Sashidhar RB. Epidemiological profile of snake-bite cases from Andhra Pradesh using immunoanalytical approach. Indian J Med Res 2007;125:661-8.

27. Kumar .P.J , Clark M.L., Clinical medicine-A text book for medical students and doctors, ELBS Saunder's pub., 2nd ed., 752 -753.

28. Laurence Benett, Clinical pharmacology, 6th ed., ELBS, Churchil Living stone Pub.

29. Lee. R.G. et al, Wintrobe's clinical hematology, 9th ed., Lee and Febiger Pub., Philadelphi, Vol 2,1311-33.

30. Mehta P.J and Sainani. G.S., Bleeding following snakebite, J. Asso. Phys. Ind, 1984,32, 389.

31. Mishra S.N. Nayak. KC, Jain, A.K et al. Profile of cardiac complications of snake bite. Indian Heat Journal, 42 (3): May 1990

32. Mitrakul.C, Juzi U and Pongrujikorn. W, Antivenom therapy in Russel's viper bite, AJCP, March 1991 ,412-417.

33. Mohapatra.B.N.et al, Coagulation disorder following viper bite in Orissa, JIMA, 1992 , Jan ; J 90 (1) :12 – 14.

34. Bawaskar HS, Bawaskar PH. Profile of snakebite envenoming in western Maharashtra, India. Trans R Soc Trop Med Hyg 2002;96:79-84.

35. Myint- Lwin, Rodney, Tun, P.E, et al. bites by Russel's vioer in Burma-. Hemostatic, vascular and renal disturbances and responses to treatment, the Lancet, Dec 1985.

36. Nigam et al, Snake bite - a clinical study, In.J.Trop. Med. Sc., 1973 , 27 , 697.

37. Nishoka and Sileire, A clinical study of 292 cases of Lance headed viper bites in a Brazilian Teaching hospital. Amer. J. Trop. Med. And Hyg. 47 (6), 1992,(805-810)

38. Paul V, Pratibha S, Prahlad KA, Earali J, Francis S, Lewis F. High-dose anti-snake venom versus low-dose anti-snake venom in the treatment of poisonous snake bites-a critical study. J Assoc Physicians India 2004;52:14-7.

39. Peter Viccellio, M.D., Hand book of toxicology, Little Brown Pub. 1993 : 685 -697.

40. Rakel, Conn's current therapy 1993, Saunder's publ., 146 - 1148.

41. Reid.H.A,Adder bites in Britain, BMJ, 1976, 2, 153 - 6.

42. Shiau DT, Sanders JW, Putnam SD, Buff A, Beasley W, Tribble DR, et al. Self-reported incidence of snake, spider, and scorpion encounters among deployed US military in Iraq and Afghanistan. Mil Med 2007;172:1099-102.

43. Punde DP. Management of snake-bite in rural Maharashtra: A 10-year experience. Natl Med J India 2005;18:71-5.

44. Sharma N, Chauhan S, Faruqi S, Bhat P, Varma S. Snake envenomation in a north Indian hospital. Emerg Med J 2005;22:118-20.

45. Reid H.A and Theakston R.D.G, Bulletin of World Health Organizational (6); 885-895 (1983).

46. Russel. E.F et al , Snake venom poisoning in the United States, JAMA, July 28 1975, Vol 233, No 4, 341 - 44.

47. Russel.F.E Snake venom poisoning. Philadelphia : Lippincott, 1980.

48. Russell, F.E. Snake venom poisoning in the United States. JAMA 1975; 233 : 341.

49. Saini. R.K, Arya, R.K, Singh., Sharma.S. et al. coagulation defects in snake bite poisoning JAPI, 33 - 2 ; 1985.

50. Sarangi. A. Jena I et al, Profile of snakebite poisoning with special reference to hematological, renal, neurological electrocardiographic abnormalities. Jr. Asso. Phys. Ind. Vol: 25 , Aug 1977.

51. Sawai, Y. and Homa,,M. Snake bite in India - Animal, plant and microbial toxins, volume 2, Plenum publishing corporation, Newyork, 451 : 1974.

52. Schwartz.S.S, Principles of surgery, McGrew Hill pub., 6th ed ., vol 1 ,183 -85

53. Scott.R.B, Price's text book of practice of medicine, 11th ed., 305-7.

54. Shankar A, Hand book of poisoning, Bhalani Pub. House, 432 - 439.

55. Sharan R, Neostigmine in the management of snake bite. JIMA., 78 ,Feb. 1982,61 - 63.

56. Silveira,P.V and Nishioka. S. South American rattle snake bite in a Brazilian teaching hospital, clinical and epidemiology study of 87 cases with analysis of factors predictive of renal failure. Trans. Royal Society of Trop. Med. And Hyg. 1992 , Sept. 86 (5); 562 - 564.

57. Stein J.H.et al, Internal medicine, Mosby pub., 4th ed., 870, 2810 - 2811.

58. TembeV.S.,Anjaria.P.D,API text book of medicine, 5th ed., API publ.,1994,1404-1406.

59. Theakstan, R.D.G. Snake venom in signs and clinical medicine. Applied immunology in snake venom research. Trans. Royal Society of Trop. Med. And Hyg. 1989 , 83 , 741 - 44.

60. Thein - Than , Tin - Tun et al, Development of renal funcion abnormalities following bites by Russel's viper in Myannar Trans. Royal society of Trop. Med. And Hyg. 1991 ; 85 : 404 - 409.

61. Tierney, Mcphee, CMDT, Apliton and Lange pub., 38th ed., 1999, 1524.

62. Virmani.S.K. and Dutt O.P, A profile of snakebite poisoning in Jammu region, J.Ind. Med. Ass., 85 ; May 1987 ; 132 - 134.

63. Warrel. D.A, Snake venoms in science and clinical medicine Russel's viper; Biology, venom and treatment of bites. Trans.Royal Societt of Trop. Med and Hyg. 83; 732 - 740 , 1989.

64. Warrel.D.A. ett al , Poisoning by bites of the saw scaledor carpet viper in Nigeria, Q.J.Med. 1977, 1977 , 47 : 33 - 62.

65. Warel. D.A, Greenwood B.M. et al , Necrosis, hemorrhage and compliment ^ depletion following the bites by the spitting cobra, Quar. J. of Med, New series, XLV, No. 177, pp 1 - 22, Jan 1976.

66. Watt. C.H, Poisonous snakebite treatment in United States, JAMA, Aug 18 , 1978 , Vol 240 , No 7 654 - 56.

67. Weatherall D.J. Ledingham. J.G and Warrell D.A, Oxford textbook of medicine, Oxford medical pub, 3rd ed., vol 1 , 1125 - 1140.

68. Weiss.H.J et al, heparin therapy in a patient bitten by a saw scaled viper, A snake whose venom activates prothromin, May 1973 , The Am.J. of Med., vol 54,653 -56

69. White.J.et al, Lymphopenia afte snake bite, The Lancet, Dec 16 1989, 1448 -9.

70. White.J, Vaughan Williams and Bruce Dunken. Lymphocytopenia after the snake bite. The Lancet ii, 1989, No 8677/ 1448.

71. William.J.W et al, textbook of hematology, McGrew Hill Pub., 4th ed., 1450 -51.

72. Wingert, W.A. Venomous snake bites. In PS Auerbach and Ec Geehr, Management of wilderness and environmental emergencies,1st ed.,Newyork;McMillan,1983; 352-378.

73. Wingert.W.A and Wainsehel. J.A quick hand book on snake bites. Med. Times 1977 ; 105 (4): 68

74. Wyhne.J. and Brondwald,E, Cardiac complications in snake bite. Cardiovascular medicine; Eugene Braunwald, W.B.Saunder's Co 1997.

75. A.K Ansari, SA Sheikh. Management of viperidae snake bite.Pak Armed Forces Med J 2000;50(1): 26–8.

PROFORMA

Sl.No	IP No	Name	Age in years	Sex	Occupation	time	season	interval in hours
1	520824	Venkatlakshmi	28	F	Agriculturist	5:30p	Rainy	4
2	560143	Hanumanthappa	70	M	Farmer	6.30p	Summer	4
3	556479	Jayamma	30	F	Agriculturist	8p	Rainy	7
4	499496	Chandrappa	55	M	Cooli	5:00 AM	Rainy	2
5	499494	Narayanapa	45	M	Farmer	6.30a	Rainy	4
6	509637	Devarappa	60	M	Farmer	8a	Rainy	3
7	478922	Munivenkatappa	55	M	Agriculturist	8;15p	Rainy	6
8	525724	Mohd Ansar	20	M	Student	9:15a	Rainy	8
9	522614	Prabhakar	35	M	Cooli	7p	Rainy	2
10	542300	Shankara	32	M	Cooli	2a	Summer	5
11	518722	Venkatesh	35	M	Farmer	11:20a	Rainy	4
12	540566	Mahesh	22	M	Student	8p	Summer	1
13	492346	Ambrish	28	M	Student	6:45p	Rainy	3
14	509124	Adamma	35	F	Agriculturist	5p	Rainy	4
15	507995	Gowramma	55	F	Farmer	7p	Summer	18
16	554902	Naraynappa	60	M	Agriculturist	5:30p	Summer	8
17	551383	Madevappa	40	M	Farmer	7:30p	Summer	2
18	551675	Narayanappa	40	M	Farmer	1:30p	Rainy	8
19	530880	Bhanuprakash	18	M	Student	10p	Summer	7
20	527969	Munikrishna	27	M	Farmer	8:15p	Rainy	4
21	512628	Seenappa	30	M	Farmer	10p	Rainy	13
22	507978	Nanamma	45	F	Agriculturist	3p	Rainy	1
23	477374	Venkateshappa	50	M	Farmer	2p	Rainy	1
24	547856	Sujatamma	38	F	Cooli	3p	Rainy	5
25	473227	Lokesh	20	M	Student	4p	Rainy	7
26	534313	Changamma	75	F	Agriculturist	5:30p	Winter	2
27	484077	Naraynappa	60	M	Farmer	7a	Winter	3
28	485604	Rathnamma	44	M	Agriculturist	7p	Rainy	4
29	522707	Seenappa	35	M	Farmer	4:50p	Summer	4
30	510341	Ravikumar	20	M	Student	4pm	Rainy	7
31	501572	Subbegowda	60	M	Farmer	8a	Winter	3
32	538046	Shobamma	30	F	Agriculturist	7:30p	Summer	3
33	514934	Nagaraj	60	M	Farmer	9a	Summer	2
34	494220	Venkatranappa	38	M	Farmer	8p	Rainy	7
35	514983	Amaramma	25	F	Farmer	8a	Rainy	5

36	510354	Gayathrama	21	F	Agriculturist	2:30a	Rainy	18
37	537124	Hairsh	20	M	Agriculturist	9:30p	Rainy	4
38	493810	Syed owais	21	M	Student	10p	Summer	3
39	556499	Ramakka	43	F	Farmer	5:30p	Rainy	4
40	533104	Savitramma	28	F	Agriculturist	4p	Summer	4
41	483078	Basavappa	56	M	Farmer	9:30a	Rainy	8
42	487212	Venkatappa	39	M	Farmer	4:30p	Summer	2
43	481788	Chinamma	40	F	Agriculturist	5:30a	Summer	4
44	521309	Ganesh	55	M	Farmer	6:30p	Summer	5
45	331259	Srinivasa	28	M	Farmer	7:30p	Summer	10
46	462140	Shantamma	36	F	House wife	11p	Winter	4
47	393210	Narayanswamy	48	M	Farmer	12:30p	Winter	9
48	282910	Seethamma	38	M	House wife	8p	Winter	3
49	349218	Muniswamy	70	F	Farmer	8p	Winter	5
50	524198	Seenappa	55	M	Farmer	9p	Rainy	18

Place of bite	First aid given	type	Site of bit	Bleeding	Vomitting	Dis coloration	Purpurae	Ptosis
Outside house	n	?	Lt foot	N	N	N	N	N
Fields	N	viper	Lt Foot	Y	Y	Y	N	N
Outside house	Y	viper	Lt Foot	Y	N	Y	N	Y
Fields	Y	viper	Lt Foot	Y	Y	N	N	N
Outside house	Y	cobra	Lt Foot	N	Y	N	N	Y
Fields	y	krait	Lt Foot	N	N	Y	N	Y
Fields	Y	Viper	Rt foot	N	N	Y	N	N
Fields	Y	?	Rt foot	Y	N	Y	N	N
Fields	Y	Viper	Lt Foot	Y	N	Y	Y	N
Fields	Y	?	Rt foot	N	N	N	N	N
Fields	Y	?	Lt Foot	N	N	N	N	N
Outside house	Y	?	Rt foot	Y	Y	Y	N	N
Fields	n	?	Lt.hand	N	N	N	N	N
Fields	N	?	Lt Foot	N	N	N	N	N
Outside house	y	cobra	Lt Foot	N	Y	N	N	N
Fields	N	cobra	Rt foot	N	N	N	N	Y
Fields	y	?	Rt foot	N	N	Y	N	N
Outside house	Y	?	Rt foot	N	N	N	N	N
Outside house	N	?	Lt Foot	N	N	N	N	N
Fields	N	cobra	Rt foot	N	N	N	N	Y
Fields	N	?	Lt Foot	N	N	N	N	N
Outside house	Y	?	Rt foot	N	N	N	N	N
Fields	Y	?	Lt Foot	N	N	N	N	N
Fields	Y	viper	Rt foot	Y	N	N	Y	N
Fields	Y	cobra	Lt Foot	N	Y	N	N	Y
Fields	N	?	Lt Foot	Y	N	N	N	N
Fields	Y	cobra	Rt hand	N	Y	N	N	Y
Fields	Y	cobra	Rt foot	N	Y	N	N	Y
Outside house	Y	?	Rt foot	N	N	N	N	N
Inside house	N	cobra	Lt.hand	N	N	N	N	N
Inside house	y	cobra	R.shoul	N	Y	N	N	Y
Inside house	N	?	R thigh	Y	N	Y	Y	N
Outside house	Y	Viper	Lt Foot	Y	Y	Y	Y	Y
Outside house	N	cobra	Lt Foot	N	Y	Y	N	Y
Fields	y	viper	Rt hand	Y	N	N	N	N

Fields	N	?	Lt Foot	N	N	N	N	N
Outside house	Y	?	Lt Foot	N	N	Y	N	N
Inside house	Y	?	Lt Foot	N	N	N	N	N
Inside house	Y	viper	Rt. buttock	Y	N	N	N	N
Outside house	Y	viper	Lt Foot	Y	N	Y	N	N
Fields	n	krait	Rt foot	y	N	Y	N	N
Fields	Y	?	Rt foot	N	N	N	N	N
Fields	Y	cobra	Rt foot	N	N	N	N	Y
Fields	Y	viper	Lt Foot	Y	N	N	N	N
Inside house	N	?	Rt foot	N	N	N	N	N
Outside house	Y	?	Lt Foot	N	N	N	N	N
Fields	Y	?	Lt Foot	Y	N	N	N	N
Fields	Y	?	Rt foot	N	N	Y	N	N
Fields	N	Viper	Lt foot	Y	N	Y	Y	N
Fields	N	viper	Lt Foot	Y	N	Y	N	Y

MASTER CHART

Diplopia/ Bluring of vision	DIFF IN swall	Giddiness	Res Paralysis	Hematuria	Gum bled	Fang marks	B.P	Pallor
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	Y	Y	Y	NOR	P
Y	N	Y	N	Y	Y	Y	NOR	AB
N	N	N	N	Y	Y	Y	NOR	P
Y	N	N	N	N	N	Y	NOR	AB
Y	N	N	N	N	N	Y	NOR	AB
N	N	Y	N	Y	Y	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	Y	Y	Y	NOR	P
N	N	N	N	N	N	Y	NOR	P
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	Y	Y	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	AB
Y	N	Y	N	N	N	Y	NOR	AB
Y	Y	N	N	N	N	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	AB
Y	N	N	N	N	N	Y	NOR	P
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	P
N	N	N	N	N	Y	Y	NOR	AB
Y	N	Y	N	N	N	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	AB
Y	N	N	N	N	N	Y	NOR	P
Y	Y	N	N	N	N	Y	NOR	P
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	P
Y	Y	Y	N	N	N	Y	NOR	AB
N	N	N	N	Y	Y	Y	NOR	AB
Y	Y	Y	N	Y	Y	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	N	Y	N	NOR	p

N	N	N	N	Y	Y	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	P
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	Y	Y	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	AB
Y	Y	N	N	N	N	Y	NOR	AB
N	N	N	N	Y	Y	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	P
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	N	N	Y	NOR	P
N	N	N	N	N	N	Y	NOR	AB
N	N	N	N	Y	Y	Y	NOR	AB
Y	Y	N	N	Y	Y	Y	NOR	P

Lymph nodes	Cr.Nr	Motor syst	Hb%	T.C	B.T-1	B.T-3	P.T-1	P.T-3	UREA	Creatinine
Absent	N	N	11	7500	2:45Min	2:45Min	14Se	14Sec	24	0.8
L.ing	N	N	6.4	13300	8”Min	5”Min	28Sec	15Sec	40	1.2
L.ing	Ptosis	N	12	5300	16”Min	8”Min	20Sec	16Sec	39	1
Absent	N	L.hemi	6.5	18000	14”min	--------	44Sec	-------	68	2.5
L.ing	ptosis	N	10	7000	3”min	3”min	13sec	13sec	20	1
R ing	N	N	13	7500	3:10Min	3:10Min	17.6sec	13.2Sec	42	1.1
Absent	N	N	11	14000	10Min	3”Min	20Sec	13Sec	22	1.1
R.ing	N	N	10	16000	1:45Min	2”Min	12Sec	12Sec	28	0.9
Absent	N	N	6.8	9000	9”Min	5”Min	32Sec	16Sec	24	0.9
R.ing	N	N	7.2	10000	3”Min	3”Min	13Sec	13Sec	18	1
Absent	N	N	13	5500	4”Min	3”Min	14Sec	13Sec	29	0.9
Absent	N	N	13	15100	17”Min	10”Min	36Sec	26Sec	107	6.8
Absent	N	N	12	6300	1:4oMin	1:40Min	12Sec	12Sec	32	0.9
L.ing	N	N	14	8000	2:15Min	2”Min	13Sec	13Sec	26	0.8
L.ing	Ptosis	N	12	12000	4:15Min	4”Min	14Sec	14Sec	40	1.4
Absent	Ptosis	N	12	12000	2:15Min	2”Min	13Sec	12Sec	20	1.2
R.ing	N	N	11	8000	2:15Min	2”Min	14Sec	14Sec	21	0.9
Absent	N	N	11	18000	4:50Min	4”Min	13Sec	14Sec	16	1
Absent	N	N	12	10600	2:10Min	2”Min	12Sec	13Sec	32	1.3
Absent	Ptosis	N	6.4	9600	2:55Min	3”Min	13Sec	12Sec	22	1
Absent	N	N	12	6000	3:18Min	3”Min	14Sec	12Sec	23	1.2
Absent	N	N	12	7600	2”Min	2”Min	14Sec	14Sec	18	1
Absent	N	N	6.3	8000	2:15Min	2:15Min	13Sec	13Sec	17	1.2
L.ing	N	N	11	20100	10”Min	8”Min	26Sec	16Sec	18	1
Absent	Ptosis	N	12	19700	4:15Min	4”Min	16Sec	12Sec	34	0.9
L.ing	N	N	13	10200	2:10Min	2:10Min	13Sec	13Sec	31	1.1
Absent	Ptosis	N	6.2	7400	4”Min	3”Min	15Sec	13Sec	17	1
Absent	Ptosis	N	6.4	16000	4”Min	3”Min	13Sec	12Sec	22	1.1
Absent	N	N	12	24700	4”Min	4”Min	14Sec	14Sec	31	1
L.axil	N	N	7.2	7000	1:45Min	2Min	13Sec	13Sec	16	0.7
Absent	Ptosis	N	11	10600	4”Min	4”Min	16Sec	12Sec	40	1.2
Absent	N	N	11	10000	5:15Min	3:15Min	26Sec	18Sec	35	0.7
L.ing	Ptosis	N	14	11000	15”Min	5”Min	26Sec	19Sec	29	1.5
L.ing	Ptosis	N	13	10400	3”Min	2”Min	14Sec	13Sec	33	0.8
R axil	N	N	6.4	9000	5”min	4”min	100sec	40sec	34	1.2

Absent	N	N	13	5300	2:45Min	2:45Min	12Sec	13Sec	21	1
L.ing	N	N	12	13200	3:30Min	3:30Min	13Sec	13Sec	37	0.9
Absent	N	N	6.9	7800	3”Min	3”Min	14Sec	14Sec	32	1.2
Absent	N	N	12	6300	3”Min	3”Min	12Sec	13Sec	18	0.8
Absent	N	N	12	8000	8:30Min	6”Min	22Sec	16Sec	22	1.3
R ing	N	N	11	1400	2“min	2”Min	13Sec	13Sec	26	1.3
R.ing	N	N	12	8000	2”Min	1:10Min	13Sec	12Sec	29	1.2
Absent	Ptosis	N	12	18000	4”Min	4”Min	13Sec	12Sec	25	1.1
Absent	N	N	13	18000	8”Min	6”Min	18Sec	14Sec	27	1.3
Absent	N	N	6.8	17200	5”Min	4”Min	13Sec	12Sec	30	1.4
Absent	N	N	12	5500	1:45Min	1:45Min	12Sec	12Sec	17	1.1
Absent	N	N	7.5	6300	1:30Min	1:40Min	13Sec	13Sec	36	1.3
R.ing	N	N	11	10000	2:10Min	2:10Min	12Sec	12Sec	43	0.7
L.ing	N	N	11	24000	8”Min	5”Min	24Sec	13Sec	34	0.8
L.ing	Ptosis	N	6.8	13300	4”Min	4”Min	13Sec	13Sec	101	3.1

E.C.G	Hematuria	Albumin	Stool	ASV	Reactions	Surgery	Saty in Days	Moratlity
N	AB	AB	N	10	NO	NIL	5	N
N	P	P	P	15	NO	NIL	5	N
N	P	P	P	40	NO	NIL	8	N
st↓	AB	AB	N	15	NO	NIL	1	MORT
N	AB	AB	N	12	NO	NIL	5	N
N	AB	AB	N	40	NO	NIL	7	N
N	AB	AB	N	27	P	NIL	6	N
N	AB	AB	N	51	NO	FASIO	8	N
N	P	P	P	24	NO	NIL	6	N
N	AB	AB	N	10	NO	NIL	2	N
N	AB	AB	N	0	NO	NIL	2	N
s.tac	P	P	P	15	NO	NIL	7	N
N	AB	AB	N	5	NO	NIL	5	N
N	AB	AB	N	25	NO	NIL	5	N
s.tac	AB	AB	N	25	NO	NIL	6	N
N	AB	AB	N	24	NO	NIL	4	N
N	AB	AB	N	15	NO	NIL	5	N
N	AB	AB	N	10	NO	NIL	1	N
N	AB	AB	N	5	P	NIL	2	N
N	AB	AB	N	10	NO	NIL	6	N
N	AB	AB	N	10	NO	NIL	2	N
N	AB	AB	N	0	NO	NIL	3	N
N	AB	AB	N	0	NO	NIL	3	N
N	P	P	P	28	NO	NIL	3	N
N	AB	AB	N	20	NO	NIL	4	N
N	AB	AB	N	9	NO	NIL	5	N
N	AB	AB	N	37	NO	NIL	5	N
N	AB	AB	N	23	NO	NIL	6	N
N	AB	AB	N	11	NO	NIL	3	N
s.tac	P	P	P	16	NO	NIL	4	N
s.tac	AB	AB	N	40	P	NIL	6	N
N	P	P	P	32	NO	NIL	5	N
N	P	P	P	26	NO	FASIO	10	N
N	AB	AB	N	20	NO	NIL	8	N
N	AB	AB	N	25	NO	NIL	6	N

s.tac	AB	AB	N	15	NO	NIL	2	N
N	AB	AB	N	25	NO	FASIO	9	N
N	AB	AB	N	0	NO	NIL	2	N
N	AB	AB	N	20	NO	NIL	4	N
N	P	P	P	32	NO	NIL	4	N
N	AB	AB	N	15	NO	NIL	8	N
N	AB	AB	N	0	NO	NIL	3	N
N	AB	AB	N	25	NO	NIL	4	N
N	P	P	P	18	NO	NIL	5	N
s.tac	AB	AB	N	5	NO	NIL	5	N
N	AB	AB	N	5	NO	NIL	2	N
N	AB	AB	N	5	NO	NIL	2	N
N	AB	AB	N	6	NO	NIL	7	N
N	P	P	P	31	NO	NIL	7	N
s.tac	AB	AB	N	30	NO	NIL	12	N