A DISSERTATION ON

"A STUDY ON PREDICTORS OF POOR OUTCOME IN SNAKE BITE INDUCED ACUTE KIDNEY INJURY AT CHENGALPATTU MEDICAL COLLEGE HOSPITAL"

Dissertation submitted to

THE TAMILNADU Dr. M.G.R MEDICALUNIVERSITY,

CHENNAI-600 032

In partial fulfilment of the regulations

For the award of the degree of

M.D (GENERAL MEDICINE)

BRANCH-1

REG.NO:200120103009

GOVT. CHENGALPATTU MEDICAL COLLEGE & HOSPITAL,

CHENGALPATTU-603001

MAY 2023

CERTIFICATE

This is to certify that the dissertation titled **"A STUDY ON PREDICTORS OF POOR OUTCOME IN SNAKE BITE INDUCED ACUTE KIDNEY INJURY AT CHENGALPATTU MEDICAL COLLEGE HOSPITAL"** is the bonafide original work of **Dr. NANDAKUMAR M** ,in partial fulfilment of the requirements for M.D. Branch-1 (General Medicine) Examination of the Tamil Nadu Dr. M.G.R. Medical University to be held in May 2023. The period of study was from April 2021 to March 2022.

12.12.22

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BONAFIDE CERTIFICATE

This is to certify that dissertation **"A STUDY ON PREDICTORS OF POOR OUTCOME IN SNAKE BITE INDUCED ACUTE KIDNEY INJURY AT CHENGALPATTU MEDICAL COLLEGE HOSPITAL"** is a bonafide work performed by **Dr. NANDAKUMAR M.,** postgraduate student of General medicine, Chengalpattu medical college, Chengalpattu, under my guidance and supervision in fulfilment of regulations of The Tamil Nadu Dr. M.G.R Medical University for the award of M.D. Degree during the Academic period 2020-2023.

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DECLARATION

I, **Dr.NANDAKUMAR M.,** solemnly declare that dissertation titled **"A STUDY ON PREDICTORS OF POOR OUTCOME IN SNAKE BITE INDUCED ACUTE KIDNEY INJURY AT CHENGALPATTU MEDICAL COLLEGE HOSPITAL"** is a bonafide record of work done by me in the Department of Internal Medicine, Government Chengalpattu Medical College and Hospital during April 2021 to March 2022 under the guidance of Prof. Dr. R. NARMADHA LAKSHMI, M.D, DCH, Professor of General Medicine, Government Chengalpattu Medical College and Hospital, Chengalpattu. This dissertation is submitted to Tamil Nadu Dr. M.G.R. Medical University, in partial fulfilment of the University regulations for the award of M.D. Degree (Branch 1) General Medicine- May 2023.

SIGNATURE OF THE CANDIDATE

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I would like to thank our beloved Dean, Govt. Chengalpattu Medical College and Hospital, Dr. **DR.K.NARAYANASAMY M.D.,D.M.,**, for permitting me to utilize the hospital facilities for this dissertation.

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This is to certify that this dissertation work titled "A STUDY ON PREDICTORS OF POOR OUTCOME IN SNAKE BITE INDUCED ACUTE KIDNEY INJURY AT CHENGALPATTU MEDICAL COLLEGE HOSPITAL" of the candidate DR.NANDAKUMAR M with the registration Number for the award of degree of M.D. General Medicine (Reg Number: 200120103009). I personally verified the urkund.com website for the plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion pages and result shows **12 percentage** of plagiarism in the dissertation.

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IEC Approval Letter – Amended 11.01.2021

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Title of Work: A Study on predictors of poor outcome in snake bite induced acute kidney injury at Chengalpattu Medical College Hospital.

The request for an approval from the Institutional Ethical Committee (IEC) was considered on the IEC e-meeting held as per ICH-GCP guidelines, New Drug and clinical trials Rule March 2019 requirements & ICMR – EC Guidance during COVID – 19 Pandemic on 25.03.2021 at the Medical Education Unit, Chengalpattu Government Medical College, Chengalpattu at 11.00 AM for the IEC-Chengalpattu Medical College members.

The Members of the committee, the Secretary and the Chairperson are pleased to inform you that your proposed project mentioned above is **Approved** in its presented form.

1. This approval is valid for three years or the duration of the project whichever is less.
2. Should inform the IEC in case of any changes in study procedure, methodology, sample size investigation, investigator or guide or any other changes.
3. Should not deviate from the area of work for which you had applied for ethical clearance.
4. Should inform the IEC immediately, in case of any adverse events or serious adverse reactions, if encountered during from study seven days.
5. Should abide to the rules and regulations of the institution(s).
6. Should complete the work within the specific period and if any extension is required, you should apply for the permission again for extension period.
7. A yearly progress report of the project has to be submitted to the IEC for review.
8. Should submit a copy of the trial work to the ethical committee on completion of the study.

(Dr.A.Anitha)
Member Secretary

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APPROVAL FROM COLLABORATING DEPARTMENT

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The Head of The Department,

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Through proper channel,

Respected sir/ Madam,

SUB: Dissertation on **" study on predictors of poor outcome in snake bite induced acute kidney injury at Chengalpattu medical college hospital"** - request for permission to do the study. Applied for approval of the project by Institutional ethics committee, CMCH, Chengalpattu- regarding.

I joined M.D GENERAL MEDICINE in the academic year 2020-2021 I am planning to do a study **" A " study on predictors of poor outcome in snake bite induced acute kidney at Chengalpattu medical college hospital** among patients taking treatment at CMCH, Chengalpattu, request you to grant me the permission to do the study.

Thanking you

23/3/21.
(M.NANDAKUMAR)

Yours faithfully,

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Through proper channel,

Respected sir/ Madam,

SUB: Dissertation on **" study on predictors of poor outcome in snake bite induced acute kidney injury at Chengalpattu medical college hospital"** - request for permission to do the study. Applied for approval of the project by Institutional ethics committee, CMCH, Chengalpattu- regarding.

For PROFESSOR AND HOD DEPARTMENT OF BIOCHEMISTRY CHENGALPATTU MEDICAL COLLEGE CHENGALPATTU

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Thanking you

M.N 23/3/24.

Yours faithfully, (M.NANDAKUMAR)

Place: Chengalpattu.

Date: 23/3/24.

From

The Professor and HOD,
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To

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I year Post graduate,
Department of General Medicine,
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Chengalpattu.

Dear Dr,

Sub: Permission – for Thesis - to utilize Central Lab services- reg.

Ref : Letter from you dated 23.03.2021

We are pleased to inform that we are willing to support your study titled **"A Study on Predictors of poor outcome in snakebite induced Acute Kidney injury at Chengalpattu Medical College Hospital"** and will be supporting with Complete hemogram examination, Biochemical and Histopathology laboratory services.

We further request you to **acknowledge Department of Pathology** in your study and also in your future publications.

This letter is itself may kindly be treated as our acceptance letter for this collaborative study and shall be submitted for ethical committee approval.

Date: 23.03.2021

Professor and HOD
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ABSTRACT

BACKGROUND: Snake bite is a significant cause of death and morbidity in tropical and subtropical countries such as the Indian subcontinent. Acute renal failure can result from the bite of a venomous snake, more common in Viper species of snakes. Globally published statistics on the incidence of acute kidney injury (AKI) after venomous snakebites in developing countries such as India are less, and the proportion of victims who rely on conventional treatments is often fatal.

AIM: To determine the incidence of acute renal injury and to examine the clinical, hemodynamic, and investigational profiles of those who were admitted with snakebite and analyzing its outcomes. To identify and categorise patients with AKI using the AKIN staging and to understand the root causes of AKI development.

MATERIALS AND METHODS: After getting approval of institutional ethical committee, 150patients who fulfilled the inclusion and exclusion criteria were recruited for the study. Details regarding initial treatment, renal parameters have been collected.Acute kidney injury was defined according to AKIN criteria. Patients were classified into 4 groups- NO AKI, AKIN 1, AKIN 2, AKIN 3 by monitoring urine output and Serum Creatinine. CBC- Hb, PCV, and Platelet count was monitored daily.Renal parameters were monitored daily. The study design was a cross sectional study. All data collected were noted using a structured proforma, including the investigations. Data was analysed using statistical package and SPSS structured software to find out the proportion of acute kidney Injury among 150 patients, and their clinical profile and outcome of them.

RESULTS: In our study, 99 patients developed AKI. 92% of patients recovered completely.64 patients with AKIN 1, 13 patients with AKIN2, 1 patients with AKIN 3 recovered with conservative management. 7 patients become Dialysis dependent for more than a month and progressed to chronic kidney Disease and advised to monitor renal parameters regularly and 5 patient died in hospital, 4 of them in stage3 AKIN and one person in stage 1 AKIN.

CONCLUSION: Our study concluded that a severe acute renal damage is highly linked with a delay in bringing the patient to the hospital. Poor prognosis is seen in patients with rapidly progressing cellulitis and those who have coagulopathy, moderate to severe thrombocytopenia also in elderly patients with co morbidities.

KEYWORDS: Snake bite, Acute kidney injury, Anti snake venom,coagulopathy.

ABBREVIATION

AKI – Acute Kidney Injury

AKIN – Acute Kidney Injury Network

ARDS – Acute Respiratory Distress Syndrome

ARF – Acute RenalFailure

ASV – Anti Snake Venom

ATN – Acute TubularNecrosis

aPTT – Activated Partial ThromboplastinTime

BT – BleedingTime

BUN – Blood Urea Nitrogen

CRF – Chronic RenalFailure

CT – Clottingtime

CT KUB – Computerized Tomography- KUB

CVP – Central VenousPressure

DIC – Disseminated IntravascularCoagulation

ECG – ElectroCardioGram

IgE – Immuno Globulin E

IV – IntraVenous

FDPs – Fibrin Degradation Products

FFP – Fresh FrozenPlasma

Hb – Hemoglobin

KUB – Kidney, Ureter, Bladder

LDH – Lactate Dehydrogenase

MOSF – Multi Organ SystemFailure

PCV – Packed CellVolume

PT – Prothrombintime

RBC – Red Blood Cell

RBS – Random BloodSugar

RIFLE – Risk, Injury, Failure and End stage Renal Disease

SAE – South East Asian Countries

USG – UltraSonoGram

WBCT – Whole Blood Clotting Time

WHO – World Health Organization.

TABLE OF CONTENTS

INTRODUCTION

Snake bite is a significant cause of death and morbidity in tropical and subtropical countries such as the Indian subcontinent. Poisoning by venomous snakes is a common occupational hazard among farmers and people living in rural and woodland areas.

India is the leading cause of earlier deaths in rural areas due to snake bite[1](Bawaskar HS).Snake bite cause two types of toxicity: **Hematotoxicity** and **neurotoxicity**.

Acute renal failure can result from the bite of a venomous snake, more common in **Viper species** of snakes. Globally published statistics on the incidence of acute kidney injury (AKI) after venomous snakebites in developing countries such as India are less, and the proportion of victims who rely on conventional treatments is often fatal.

The global annual mortality rate from snakebites is estimated to about 94 000 per year, with about 15 000 deaths per year in India alone [2]

The incidence of acute kidney injury in India is **20-32%** after a viper bite. There is widespread disagreement with the available data on the incidence of acute kidney injury from snakebites[3](Warrel DA).

The specific treatment for snake envenomation is the prompt delivery of anti-snake venom. Early neutralisation of the venom's effects would result in lesser morbidity or mortality.

More than 200 of the world's 3000 snake species are located in India, and they range in length from 100mm-long worm snakes to 6m-long pythons. Snakes can be found in a variety of habitats, including marshes, lakes, farmlands, deserts, tropical oceans, and mountains. These predators are crucial in maintaining the ecology.

AIM AND OBJECTIVE:

This is a **cross sectional study** of snakebite, and the aims of the study are

1) To determine the **incidence of acute renal injury** and to examine the clinical, hemodynamic, and investigational profiles of those who were admitted with snakebite.
2) Analyzing patient outcomes in relation to various parameters such as the amount of Inj.ASV given to the patient, the bite and needle time ,comorbidities, thrombocytopenia, cellulities, and lymphadenopathy.
3) To determine whether there is a preference for the sex or age of the patient for severe envenomation.
4) To identify and categorise patients with AKI using the AKIN staging and to understand the root causes of AKI development.
5) To study the results of patients with AKI treated with various managements. Study was done in 150 Patients admitted in different medical wards in Chengalpattu medical college hospital.

REVIEW OF LITERATURE:

HISTORICAL ASPECTS:

Snakes are elongated, legless, carnivorous reptiles that do not have external ears or eyelids and belong to the **suborder Serpentines.** Their apparent rebirth through skin casting and some of their ability to cause pain and death has endowed them with a mystical aura that make some ethnic groups consider them deserving of adoration.

In the past Aesculapius said that snakes would "crawl across the bodies of sick people and lick them back to health " in ancient Greece.In India, treating a snake bite with a variety of brutal techniques, including local cuts, pricks, punctures, and attempts to suction the venomous bite mark. Use of chemicals and natural remedies to the bite mark and the use of snake stones. The key development in the management of snake envenomation was the creation of snake anti venom.The dish was made by **Albert Calmette** when his hamlet flooded in 1890 and cobras murdered more than 100 people, forty inhabitants of his village. So he captured a few snakes and milked them. He used his horses to produce antibodies. Antivenom for Indian cobras proved effective against South African cobras**.** In India, Lamb and his successors developed antivenom for the **Russell's viper.**

The polyvalent then appeared as antivenom from Kasauli, the main research facility. A Later Supportive measures including dialysis and blood transfusions were developed . Recent developments include quick immunodiagnosis and administering

particular monovalent anti-venom, creating development of a vaccination against snakes that are commonly observed nearby[4].

EPIDEMIOLOGY:

Snakes often don't prey on people. Most snakes tended to stay away from people unless they were frightened or hurt. In impoverished rural areas, snakebite is one of the most ignored public health problems particularly in communities residing in tropical regions[5](Naing S).

The majority of snake bites happen on the lower limbs of rural residents, including children and agricultural labourers. Since the majority of cases are in poor nations like India which was initially treated by licenced snake charmers, healers, and religious males who apply snake stone, chant mantras, and employ herbal treatments all of which are believed to magically suck the venom out of the wound .

In India, 46000 (99% CI 41000-51000) persons pass away from annual snakebite depending on verbal autopsy of all deaths in 6671 randomly chosen sample areas throughout the country[6](BANERJEE R)- **(Mohapatra B 2011).**0.5% of all deaths were caused by snake bite, and 97% of victims perished in rural regions with only 23% receiving medical attention.

Krait bite occurs during the hours of 12am and 4am, therefore patients will develop neurotoxicity and will be unconscious due to the bite and may be misdiagnosed as cerebrovascular accident in the morning [7](Reid HA.).

In rural locations, victims frequently misidentify the snake's species or fail to discover it altogether, especially when it's a cobra. This frequently occurs with kids. Due to inadequate snake knowledge, inadequate visibility, fear of snakes, and other factors may result in inaccurate identification of snakes[8](Saini RK,)

SNAKE BITE IN INDIA:

The "**Million Death Study**," conducted by the Registrar-General of India from 2001 to 2003,predicted to deliver trustworthy proof of significant mortality (It is based on **Representative, Re-sampled, Regular (RRR) data** (50 000 per year).Hati et al. Conducted a sizable field survey in 1992 in West Bengal's Burdwan district villages disclosed every year 8000 people were bitten by snakes, among them 800 peoples died[9](Hati A.K.).

SNAKES:

Only 667 of the 3346 snake species documented worldwide have ventral fangs that allow them to inject venom when biting people or other animals. There are around **242 different species** of snake in India.**57 are lethal**and are members of the **Colubridae, Eliapidae, and Viperidae.**

Viperid, Crotalid, and Colubrid bites typically have abnormalities in hemostasis and signs of local envenomation. The Elapidae frequently causes neurological symptoms and paralysis whereas rhabdomyolysis is caused by hydrophidae . The **saw or carpet Echis carinatus, a scaled viper**, is legitimately one of the most dangerous snakes[7](manson)

VENOMOUS SNAKES:

There are three families of venomous snakes.

The family **ATRACTASPIDINAE** includes adders and false vipers. Extremely lengthy **solenoglyphic** (hinged erectile fangs). Also known as a stiletto or side stabbing snakes, as they swat at their victims from the side and the corner of the partially closed lips is showing fangs.

Short fixed proteoglypous family: **ELAPIDAE (short fixed fangs).** This comprises Sea snakes, coral snakes, mambas, cobras, and kraits.

Old world vipers and pit vipers are members of the **VIPERIDAE** family.Vipers have long, curved, hinged fangs with a closed venom duct in the front. Pit vipers can recognise warm-blooded animals- thanks to an infrared heat-sensitive organ.

NON VENOMOUS SNAKE:

BORDAE FAMILY: Known as the enormous constrictors and notorious for suffocating their prey. Examples are Australian serous Pythons, South American anacondas, African rock pythons, and reticulated pythons.

The largest family of snakes, **COLUSRIDAE**, contains 1748 species.The amount of venom, a snake on its first bite and the lethal dose vary between snakes for men. Sometimes only 1/6 of the venom is used and is deadly to a healthy person.

THE BIG FOUR SNAKES

WHO - MEDICALLY IMPORTANT SNAKES IN INDIA

Category 1:	**Elapidae:** *Bungarus caeruleus; Naja kaouthia* (north-east), *Naja naja* (throughout) **Viperidae:** *Daboia russelii, Echis carinatus; Hypnale hypnale* (south-west)
Category 2:	**Elapidae:** *Bungarus fasciatus, Bungarus niger, Bungarus sindanus, Bungarus walli; Naja oxiana* (north-west), *Naja sagittifera* (Andaman Islands); *Ophiophagus hannah* (south, north-east, Andaman Islands); **Viperidae:** *Cryptelytrops albolabris, Cryptelytrops purpureomaculatus* (east), *Trimeresurus malabaricus* (south-west), *Trimeresurus gramineus* (south India, Andaman & Nicobar Islands), *Macrovipera lebetina* (north-west).

COMMON COBRA :

Zoological Name : NajaNaja

Family : Elapidae

Location : Commonly seen in India, Pakistan, Bangladesh, Nepal.

In India, it is abundantly seen in Assam & Kashmir.

Tamil Name : நல்லபாம்பு/நாகப்பாம்பு

Appearance : Scales on the ventricles might be grey, yellow, black, or brown.

A hood mark or occasionally salt-and-pepper speckles can be spotted on the dorsal scales. Two circular ocelli patterns become joined when the hood is present by what looks like a spectacle-like curved line. It is one to five metres long.

Identifying Characteristics:

When frightened, it stretches the head and develops hood. A mark in the shape of a spectacle will be found on the dorsal side of the hood.

INDIAN KRAIT (BLUE KRAIT) :

Zoological Name:BungarusCaeruleus

Family : Elapidae

Tamil Name : கட்டுவிரியன்

Location: Commonly seen in south India & Sri Lanka.

IMAGES OF ELAPIDAE SPECIES

Figure 2.1. SPECTACLED COBRA WITH HOOD

Figure 2.2. COBRA WITH SPECTACLE

Figure 2.3 DORSAL SURFACE OF KRAIT

Figure 2.3. VENTRAL ASPECT OF INDIAN KRAIT

APPEARANCE :

Flat head and little perceptible neck. The head shields are standard and unlaureled. It typically has white crossbars on the dorsal side and is black in colour. The snake's ventral region, or belly, is unadulterated.

BEHAVIOUR:

It is slow and normally inert during the day, but at night it is active and hisses loudly . It will create coils when agitated and bury its head and becomes flattened.

RUSSELL'S VIPER (CHAIN VIPER / SEVEN PACER / SCISSOR SNAKE) :

Zoological Name: DabioaRusselli

Family : Viperidae

Location: Commonly seen in India, Pakistan, Sri Lanka, Bangladesh, Nepal, and Thailand.

Tamil Name :கண்ணாடிவிரியன்

APPEARANCE:

It has a triangular head, and its crown is covered in scales that are highly irregularly fractured. The ventral scales range in number from 153 to 180, while the dorsal scales are strongly keeled. The head features two distinctive dark spot with a brownish V or X marking.The maxillary bones can support two to six pairs of fangs at once, with the first pair being the active pair and the others serving as backups. The maximum length of the snake is about 5.5 feet tall.It is a nocturnal forager, however in the winter it changes its habits and starts to function during the day. When threatened, it creates a succession of S shapes loops and lift the body's proximal third and emit a hiss.

SAW SCALED VIPER:

Zoological Name:Echiscarinatus.

Family : Viperidae

Tamil Name : சுருட்டைவிரியன்

Location : India, Pakistan, Sri Lanka, Bangladesh.

APPEARANCE:

The head is distinct from the neck, which is covered with dorsally keeled and ventrally rounded scales. Pale buff, greyish, or reddish with a succession of spots that are different colours with a lighter interblotch Patches.

BEHAVIOR:

It is primarily nocturnal and crepuscular. Active primarily after rains or during muggy evenings. A double coil and a figure of eight are its standard position.Head in the middle, allowing it to lash out like a freed animal.

POISONOUS SPECIES OF VIPERIDAE FAMILY

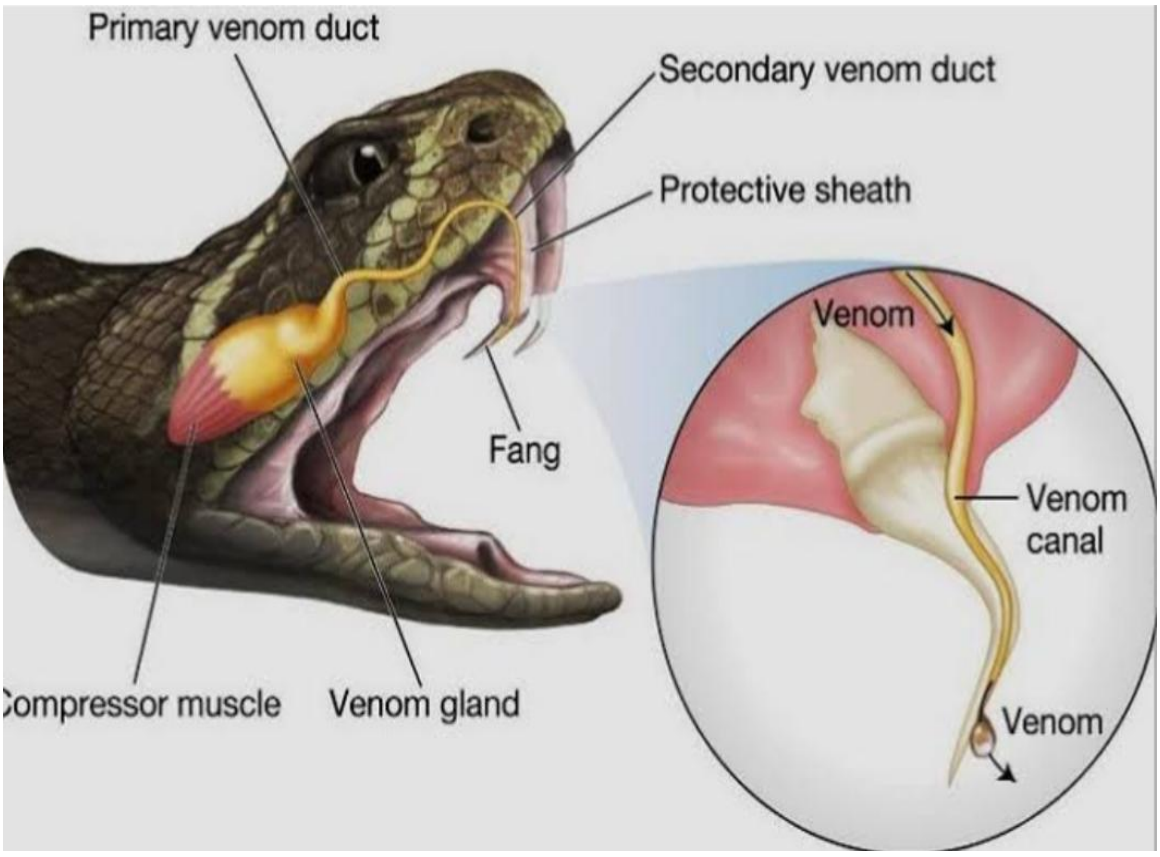
Figure 3.1 Dorsal aspect of Russell viper

Figure 3.2 Ventral aspect of Russell Viper

Figure 3.3. SAW SCALED VIPER

SNAKE VENOM:

Venom from snakes is protein-based and comprises a variety of polypeptides, enzymes, and coagulant and anticoagulant compounds. It is a rich bio-resource of biologically active substances. Each snake's venom contains specific chemicals that are essential not only to comprehend the pathophysiological changes but also to guide and prepare therapy for snake bites [(11)](Sharma N, Chauhan S).



NATURE OF VENOM:

PHYSICAL APPEARANCE:

A clear, translucent, light yellow or straw-coloured viscous fluid characterises snake venom. It has a specific gravity of 1.03 to 1.07 and is thermo-labile. Varied species venoms have different pH, depending on when they are produced.

When they are exposed to light, they become slightly turbid[12](BROOK V. B. 1971).It can be crystallised when dried. Venom that has been dried and lyophilized is thermostable. It is soluble in water and glycerine. Destroyed by $KMnO_4$, $AgNO_3$, as well as alcohol and strong alkalies like NaOH and KOH, etc

BIOCHEMICAL COMPOSITION OF SNAKE VENOM:

The amounts of enzymes, non-enzymatic poisons, non-toxic proteins, and acetyl choline in snake venom vary.

Enzymes: About 10–90% of viperid and 25–75% of elapid venoms are made up of enzymes. Various snake enzymes are discussed below,

Proteinases: They significantly destroy tissue due to the digestion of peptides and tissue proteins.The endothelium-damaged may then cause the breakdown of arteries and veins.

Hyaluronidases: These enzymes help in rapid diffusion of venom from the site of bite by helping in dissolution of hyaluronic acid in connective tissue.

Acetyl Choline Esterase: This enzyme, which is mostly found in elapid venom, helps break down acetyl choline into acetic acid and choline, impairing neuromuscular transmission.

Phospholipase: Phospholipase A2 directly hydrolyzes the phospholipid cell membrane and generates hemolytic substances such as lysolecithin,thus encouraging hemolysis.

Phosphodiesterase: It targets the derivatives of DNA, RNA, and nucleotides.The decrease in systemic arterial pressure is caused by this.

Acetyl choline: It directly affects the heart or the neuromuscular system.

Metalloproteinases: It cause endothelial lining disturbance and it is the primary reason for cellulitis.

PROCOAGULANT ACTIVITIES: Numerous procoagulants found in snake venom interact with one another at the coagulation cascade to activate the coagulation mechanisms that lead to the development of microthrombi (a mechanism similar to DIC) and consumption.Coagulation disorder, which accounts for snakes longer clotting times.

Direct prothrombin activator: This chemical, which is nameless yet directly stimulates the prothrombin with no need for activation, cleaves one or more peptide bonds in human and bovine tissue which produce an intermediate that is catalytically active and it automatically transforms into a thrombin.Additionally, this mechanism is shown in Elapid, Viperid, and Colubride.

Activator of factor V: Russell Viper venom has a particular peptide that activates Factor V and triggers the clotting cascade.

Activator of factor X: Macfarlane learned that a particular chemical contained in Russell viper venom when calcium is present can activatesFactor X, andcauses Factor Xto alter in conformation.

Activator of Factor IX: The Russell Viper's Factor X activator also stimulates Factor IX.

Prothrombin indirect activator: This factor along with factor X convert prothrombin to thrombin.

Thrombin like peptides: Venom of vipiridea contains glycopeptides that are not inhibited by heparin , and can indirectly activate thrombin.

ANTICOAGULANT ACTIVITIES:

In addition to pro-coagulants, snake venom also contains anti-coagulants, which produce anticoagulation through the following mechanisms:

1)Preventing activation or inhibiting one or more coagulation factors.

2)Directly causes fibrinolysis by activating fibrinogen.

3)Direct plasminogen activation, which brings about fibrinolysis.

NON ENZYMATIC POLYPEPTIDE TOXIN PROTEIN:

In general, neurotoxins and hemotoxins are the two kinds of toxins found in snake venom, however there are also myotoxins and cardiotoxins. No snake venom has just one kind of toxin. All Possess a mixture of toxins.

HEMOTOXINS:

Toxin causes major injury to internal organs and other tissues by lysing erythrocytes and interfering with blood coagulation processes. These toxins typically work more slower than neurotoxins.

CARDIOTOXIN:

These bind particularly to cardiac cells, which causes an increase in cellular depolarization and an inhibition of heart contractility. On occasion, the toxins cause the membrane's surface to leak calcium. Cardiac arrest can result from this mechanism. Some chemicals cause capillary leak syndrome that results in third space fluid and intravascular hypovolemia, all of which produced shock.

NEUROTOXINS:

Low molecular weight neurotoxins are present in Elapidae species, as well as in the venoms of Hydrophid, Vipridae, and Crotalid species. There are two types of neurotoxins and both are postsynaptically acting.

TOXINS AFTER SYNAPTIC BLOCKING:

Cobratoxins and alpha-Bungarotoxins are examples of alpha-neurotoxins.They bind to the cholinergic Nicotinic Acetylcholine Receptors neurons found in skeletal muscle's motor end-plates because they imitate the acetylcholine molecule's structure, its ability to attach to receptors, and restrict the passage of acetylcholine, causes numbness and paralysis.

PRESYNAPTIC NEUROTOXIN:

Beta-neurotoxins, also known as presynaptic phospholipases A2, include crotoxin, taipoxin, and beta-bungarotoxins. Through preventing the release of acetylcholine from the presynaptic membrane ,they may results in flaccid paralysis.

FASCICULINS:

These neurotoxins act synergistically to block acetylcholine esterases, which results in buildup of Ach ,this condition is serious, widespread, and persistent fasciculations (rapid muscular contractions) and could result in mortality.It is primarily present in mambas and a few rattlesnakes.

DENDROTOXINS:

Dendrotoxins are synergistic toxins that bind to voltage-gated potassium channels and impede neurotransmissions. Positive and negative ion exchange defect across the neuronal membrane results in failure to conduct nerve impulse, causing neuromuscular palsy.Specifically, bungarotoxins bind to some types of nicotinic acetylcholine receptors in several ganglia of the brain.

CYTOLYSINS:

It results in edema by lysing blood and tissue cell structures.Bleb development, necrosis, and cellulitis can occur,

SYMPTOMATOLOGY:

GENERAL SYMPTOMS:

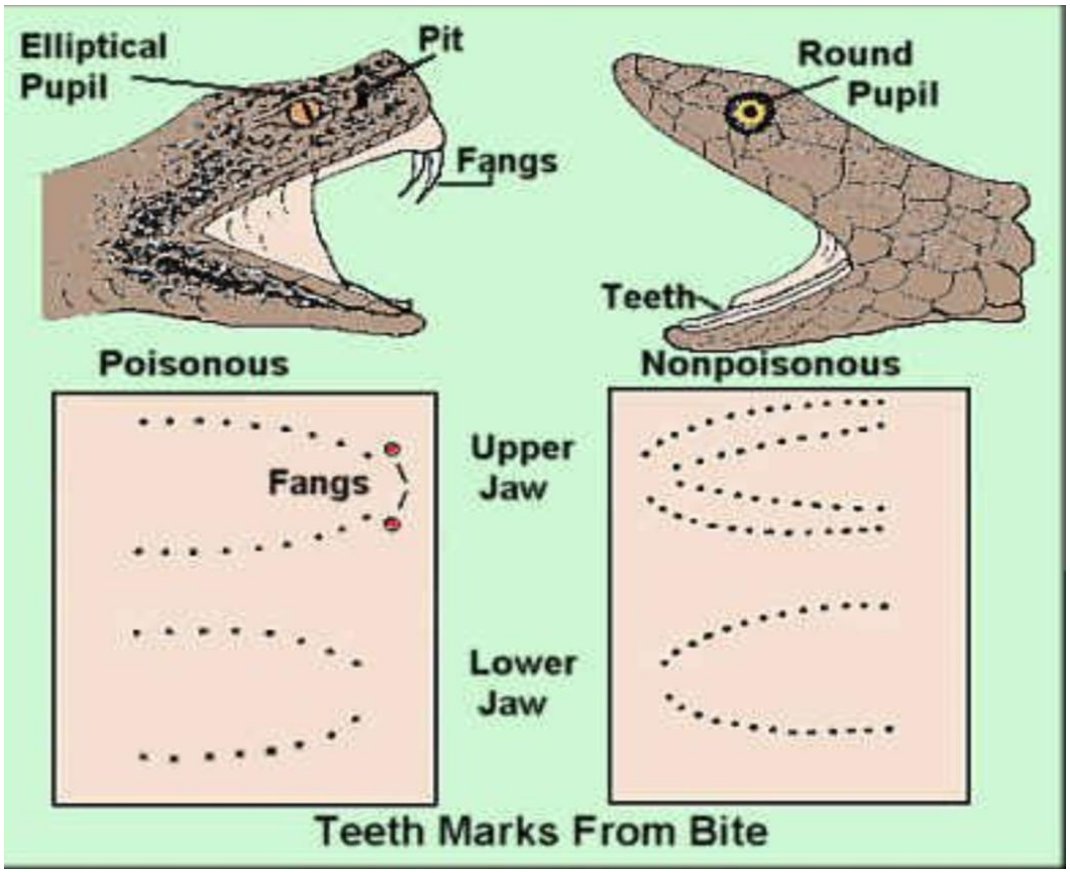
SYMPTOMS NOT RELATED TO ENVENOMATION:

Fear, anxiety, palpitations, and syncopal attacks will emerge in snake bite victims as they consider their mortality or the effects of envenomation.

Some of the patients may have increased anxiety, hyperventilation, and perspiration that could be mistaken for neurotoxic systemic symptoms. Native treatment and irrational care from traditional healers or relatives, such as cutting over the bite mark, using irritants, applying plant juices, etc produce illogical symptoms and signs that could lead to a false diagnosis. Other typical signs include headache, nausea, vomiting, and stomach pain, fatigue, sleepiness, and colic.

LOCAL SYMPTOMS:

1. Pain at the bite site
2. snake bite or fang marks noted
3. swelling
4. Fang marks that are bleeding
5. Bruising
6. Lymphadenopathy
7. Bullae formation
8. Gangrene, necrosis, local site infection.



SYSTEMIC SYMPTOMS:

Systemic symptoms are dependent on the type of snake that bites as well as the quantity and intensity of venom administered.

In majority of the time, with the symptoms and the patient's description, a snake species can be identified.

VIPERID ENVENOMATION:

Local envenomation:

1)Pain:

If the snake did not inject venom in the case of a dry bite or non-poisonous snake, the fang will pierce the skin and subcutaneous tissue during the bite, causing discomfort that may at first go away.In addition to being irritating, the venom contains several proteases.

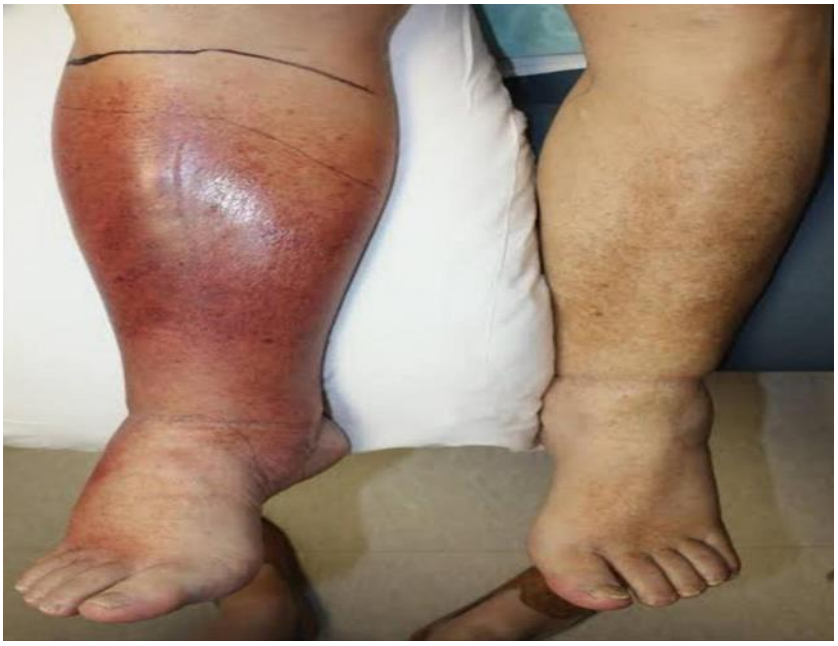
Inflammation and tissue damage caused by metalloproteinases will cause discomfort. Lymphangitis, ascending cellulitis, fluid accumulation, vascular obstructions, subsequent bacterial infections , gangrene development, ischemia, and compartment syndrome are the causes of snake bite envenomation ongoing pain.

2)SWELLING:

Viper venom will have a serious local impact. The onset of swelling typically takes 15 minutes, but very rarely takes several hours. It spreads quickly, sometimes encompassing the entire limb and nearby trunk. If the swelling is quickly changing it can be assumed to be a viper if the snake was not identified.

Bruising is frequent, especially along the route of the superficial lymphatics and over the local lymph nodes. Large volumes of blood that have been extravasated can fit in swollen limbs, causes hypovolemic shock.The localised swelling around the bite mark caused by vasculotoxic is due to penetration of the superficial tissue by the venom. Metalloproteinase , toxic polypeptides ,Phospholipases and endogenous

agonists like serotonin and histamine and kilims are the causative factors.Skin will be stretched and glossy due to edema. It is tender upon palpation, and the swelling may extend centripetally to groin from foot.



CELLULITIS RIGHT LEG

3)ECCHYMOSIS:

A few hours after the bite, the skin above the swelling area begins to discolour. It depends on the amount of venom administered and the species of snake bite.

4)FORMATION OF BULLAE:

Bullae will develop after a viper bite within eight hours, and it depends on the strength and volume of injected venom. The bullae will be filled with clear, serous fluid if the envenomation is moderate. If there has been a serious envenomation hemorrhagic bullae will develop, frequently burst, and cause ulcers.[13](REID H. A. 1963)

5)GANGRENE AND NECROSIS:

Bite at sites of tight facial compartment like anterior part of tibial portion can result in cellulitis and high intra compartmental pressure causing ischemia and necrosis of the surrounding tissue acting along with the toxins.

6)SYSTEMIC MANIFESTATIONS:

CIRCULATORY SHOCK:

The venom's peptide and protein composition activates the kinin system, which is then inhibited by the bradykinin system. One of the main causes of death from viper bites is shock. It may either due to hypovolemia due to hemorrhage.

A swollen limb can hold many litres of extravasated blood, which can result in hypovolemic shock, which can occur 6 to 26 hours after the initial envenomation. Hemolysis combined with renal and respiratory failure makes shock much worse.This can happen immediately after the bite or three to four days afterwards.

HEMOSTATIC ABNORMALITIES:

A major contributor in bleeding caused by the consumption of coagulation factors is factor XII, which is specifically activated by hemorrhagins, a special toxic component of viper venom. Fibrinogenolysis, a different process, has been proposed as a Subsequent haemorrhage from Echiscarinatus bites.[14](V.N.Acharya 1981). R.K. Sainiof SMGS Hospital and government medical college, Jammu, who Studied 150 cases of viper bites, found that 92% had evidence of primary fibrinogenolysis whereas only 8% had DIC

The gingival sulci is the frequent site of spontaneous hemorrhage and epistaxis can occur. A true hemoptysis is uncommon. Some people may experience haematuria.

Hours following the bite,spontaneous bleeding from the subconjuctival ,gastrointestinal, and genitourinary tracts may occurs.

Increased discoid and follicular haemorrhages, as well as petechiae occurs. The possibility of anterior pituitary bleeding (similar to Sheehan's condition) occurs.

Russell's viper envenoming can be challenging. Ongoing bleeding (more than 10 Minutes) from the the fang punctures can occur .Venepuncture site bleed are the first clinical sign of consumption coagulopathy .

Development of hemorrhagic blebs and uncontrolled bleeding from the area of bite may be the first clinical manifestation of viper envenomation.Within 2 to 24 hours of the bite, patients appear with ecchymosis, purpura, and hematoma: similar to Schwartzman phenomena.

The most typical signs of hematotoxity include hematuria, hemetemesis, malena, gum bleed, and hemoptysis.There can also be cerebral haemorrhage.

Acute renal failure can result from shock, hypovolemia, and microthrombi occluding the renal vasculature. Hemoglobinemia and hemoglobinuria are the symptoms of intravascular hemolysis.Peripheral smear can show fragmented erythrocytes.Severe anaemia and abrupt renal failure are also present in microangiopathic hemolysis.

CARDIOVASCULAR MANIFESTATION:

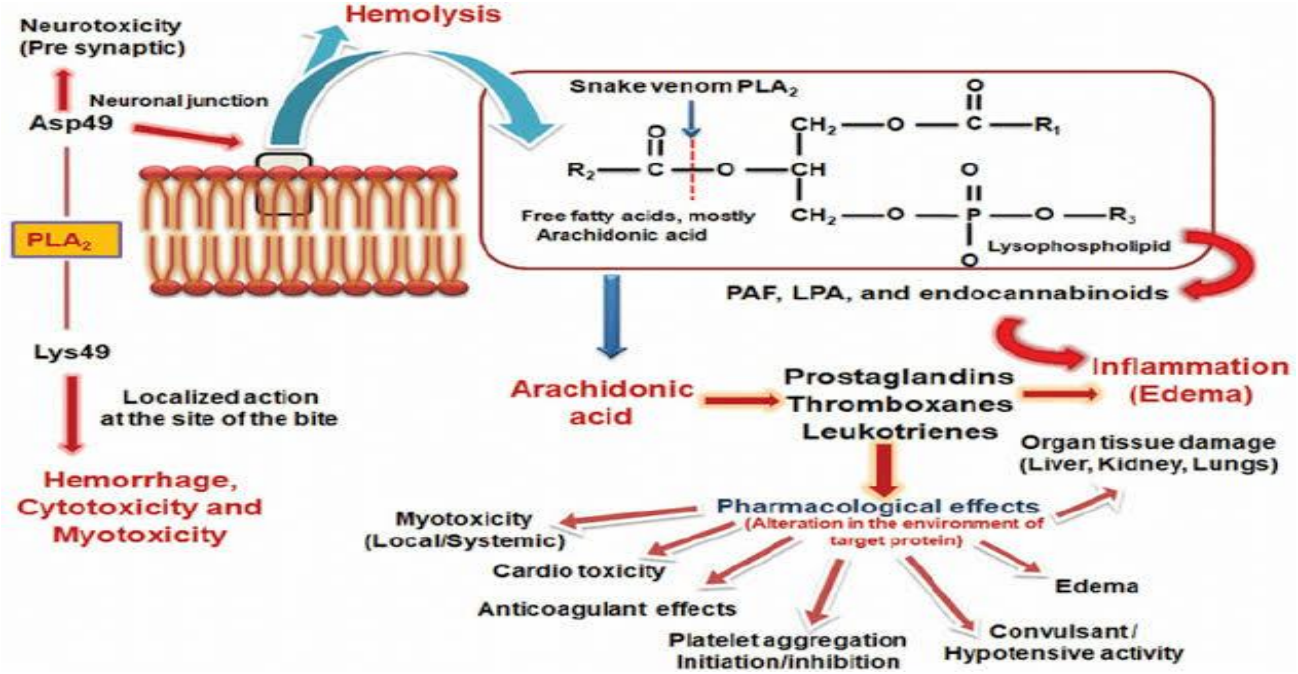
Viper bite can cause collapse, hypotension, shock, pulmonary edema, chemosis, and arrhythmias.

NEUROLOGICAL MANIFESTATIONS:

The most common symptoms include drowsiness, altered olfaction, ptosis, external opthalmoplegia, facial muscle paralysis, nasal twang of voice, aphonia, nasal regurgitation, dysphagia, respiratory paralysis, and abrupt flaccid paralysis

Venom phospholipase A2 is typically responsible for viperid neurotoxicity; nevertheless,Is a characteristic of envenoming caused by the tiny south The bitis species of Africa, the Russell's vipers of India and Sri Lanka(DusotaRussellis). Elapid envenoming brings about paralysis to respiratory muscles causing respiratory failure.Muscle soreness and widespread myalgia are symptoms of rhabdomyolysis.

FACTORS MEDIATING SNAKE BITE ENVENOMATION



ELAPID ENVENOMATION:

LOCAL SIGNS OF ENVENOMATION:

Krait typically has no local envenoming effect.A cobra bite might appear with intense local scorching pain.Cobra venom's direct cytolytic effect causes local necrosis . However it is minimal in King cobra (Ophiophagus Hannah) bites and it causes leg swelling and bullae formation.

NEUROTOXIC SYMPTOMS:

Repeated vomiting is the initial indicator of systemic envenomation, and the earliest symptom of neurotoxicity is drowsiness, which appears between 15 minutes and 5 hours after envenomation.However, bilateral ptosis is the most prevalent and early sign.

Patients report visual problems, double vision, and muscle immobility.These are crucial because they are the only ones that are evidence of mild envenomation .

Other preparalytic symptoms include hazy vision, paraesthesia, and loss of coordination, altered gustatory and olfactory perception, hyperacusis, dizziness, headache, and other autonomic signs like gooseflesh, conjunctival congestion and increased salivation occurs.

The aforementioned preparalytic symptoms all appear between one and four hours after the bite.Ptosis and external opthalmoplegia are the first symptoms of paralysis to appear.

It may occur as soon as15 minutes or as late as 10 hours following the bite.The paralysis of the vocal cords, tongue, and facial muscles followed by Neck muscles and deglutition muscles.

Depending on the level of envenomation, pupils may dilate.The symptoms may appear slowly over time or suddenly, depending on the severity of the envenomation.The patient struggles to speak clearly and has a nasal twang as a result of palatal palsy, which causes difficulty opening the mouth and swallowing. Intercostal muscle paralysis can cause a variety of symptoms, including reduced rib movement outward. The breathing shifts to a diaphragmatic pattern, then entire paralysis.

The symptoms of respiratory failure include cyanoses and excessive sweating. During this stage patient general condition worsens and pupils are non reactive , deepening of coma, twitching, convulsions and can result in death.Limb weakness usually appears last.Proximal muscles are most often affected resulting in flaccid quadriparesis.The progression of symptoms from ptosis to the development of respiratory failure is remarkably similar to that of myasthenia gravis.

HEMOTOXIC SYMPTOMS:

Krait do not typically cause coagulopathy, but Asian cobra bites does. Abnormal Coagulation mechanism is comparable to that of viperid envenomation.On the other hand, internal bleeding, intravascular hemolysis, and acute kidney injuries in elapid species are rare.

CARDIOTOXICITY:

Cardiotoxins might cause sweating, numbness in the extremities, hypotension, tachycardia, and ST-segment T wave alterations,which subsequently continues to decline, resulting in hypotension, arrhythmias, and heart failure.

MANAGEMENT OF SNAKE BITE:

1. First Aid
2. Shift the patient to the nearby hospital
3. Resuscitation
4. History and Clinical examination
5. Investigations
6. ASV treatment
7. Supportive Management
8. Treatment of complications like Respiratory Failure, Acute kidney Injury
9. Treatment for the local wounds
10. Rehabilitation

FIRST AID TREATMENT:

It is the patient's initial care provided by others or even by himself. It is crucial in the management measures because it prevents the venom from being absorbed right away.

DO'S AND DON'TS IN FIRST AID:

1) Relieve the anxiety .
2) Imobilize the limb of the patient.
3) If the required tools are available, pressure-immobilization or a pressure pad should be considered unless an elapid bite can be ruled out.
4) Avoid native treatment with the bite wound in any way (cutting, rubbing,forceful washing, massage, use of herbs or chemicals, as well as this could increase venom absorption, spread infection, and to exacerbate local bleeding [15](Baglin T).

RELEASE OF TIGHT BANDAGES:

Use of tourniquets is not advised. If used, a quick release of venom into the bloodstream could result in severe coagulopathy, abrupt hypotension, and other problems.Ideally, this shouldn't be released until the sufferer is receiving medical attention, hospital treatment, resuscitation equipment, and antivenom are all accessible and the process of treatment has begun .

HARMFUL METHODS OF FIRST AID TREATMENT:

1) Incising the bite mark should not be done.

2) Sucking the venom if harmful.

3) It was thought that placing tourniquets—tight bands—just above the place that had been bitten would prevent venom from entering the systemic circulation. Some chemicals and plant juices applied to the bite mark are done by Rural residents as

they firmly believe in conventional medical practises, hence it will postpone specific treatments, and occasionally it will confuse with local poisonous symptoms, and occasionally in cases of coagulopathy as it causes continuous bleeding from the incision site.

TRANSPORT TO HOSPITAL:

The victim should be transported to nearby Government Hospital or healthcare facility immediately .He can be shifted by using motor vehicles, bicycle, train in resting position; if possible should be transported by using Ambulance.

INITIAL RESUSCITATION:

"ABCDE APPROACH"

1. Airway patency
2. Breathing (respiratory movements)
3. Circulation (arterial pulse)
4. Disability of the nervous system (level of consciousness)
5. Exposure and environmental control (protect from cold, risk of drowning etc.)

HISTORY AND PHYSICAL EXAMINATION:

HISTORY:

To determine the severity of envenomation and determine the species of the snake that bit you, it's crucial to have a brief history of the circumstances of the bite and the development of local and systemic symptoms and indicators.

There are four significant inquiries to make:

1)Which area of your body have you been bitten in?

2)"When and how were you bit?" is the second question.

3)What is the snake that bit you ?

4)"How do you feel right now?"

Identification of species is crucial to foresee problems.

Vomiting and abdominal pain are the early signs of systemic envenomation. Some patients may also experience headaches. Record any symptoms, including bleeding, diarrhoea and double vision.Early indicators that a patient has severe envenoming include the identification of a particularly hazardous snake.A local swelling that spreads quickly and early from the biting site.

Early painful swelling of the neighbourhood lymph nodes, a sign of venom spread throughout the lymphatic system.

Early signs of a systemic condition include shock, collapse, nausea, and vomiting, strong headache, “heaviness” of the eyelids, diarrhoea, and early ophthalmoplegia or (pathological) ptosis.

Early-spontaneous systemic bleeding. Passage of dark brown/black urine.

PHYSICAL EXAMINATION:

EXAMINING THE DAMAGED PART:

Bite site:

Venomous snakes often only have one fang and leave one mark. Unless it bit more than once, which is rare. A number of scratchy marks are seen which is an essential feature to distinguish the venomous from non venomous snakes ,as most non venomous snakes have multiple fang marks . “The existence of lone fang marks, when associated with venomous snake bite showed a 100% sensitivity, a 56% specificity, and a 100% accuracy. Predictability of 89%. The discovery of several scratch scars like teeth had a 100% accuracy rate for predicting nonvenomous snakes bite[(16)]”(Paul VK).

Recordings should be made of the extent of the swelling, skin colour, the presence of bullae, and vesicles. Swelling should be marked to upper level and progression recorded. Lymph nodes may be palpated which is mostly tender.

GENERAL EXAMINATION:

In cases of neurotoxic snake bite, and patients with shock and respiratory failure, consciousness and orientation will be compromised .Pallor is a sign of internal bleeding and hidden gastrointestinal haemorrhage.

Patients who present respiratory failure may have cyanosis. Vital indicators like blood pressure, heart rate, and respiration rate should be watched for orthostatic hypotension; this could be an early warning sign of hypovolemia .Body's entire surface area should be examined for ecchymosis, subconjunctival bleeding .Internal capillary leak syndrome may cause internal abdominal bleeding and pain. Renal ischemia may be the cause of flank tenderness; it happens in envenomation by vipers.

NEUROTOXIC BULBAR PARALYSIS:

Ptosis and perioral numbness are the earliest symptoms. Check the movements of the extraocular muscles. A early warning indication of respiratory paralysis is weak neck muscles and hence ought to be examined periodically. Swallowing difficulties, nasal regurgitation, and nasal speech twang are indications of bulbar weakening, which are typical of envenomation by Elapidae. When breathing paradoxically, the abdomen expands rather than the chest on attempted inspiration" and this will happen as a result of subcostal muscle paralysis that move the diaphragm.The respiratory muscle can be evaluated with a single breath count, but if the patient was stressed, it became less valid.

Feature	Cobras	Kraits	Russell's Viper	Saw Scaled Viper	Hump Nosed Viper
Local pain/Tissue Damage	YES	NO	YES	YES	YES
Ptosis / Neurological Signs	YES	YES	YES!	NO	NO
Haemostatic abnormalities	NO*	NO!	YES	YES	YES
Renal Complications	NO	NO*	YES	NO*	YES
Response to Neostigmine	YES	NO?	NO?	NOT applicable	NOT applicable
Response to ASV	YES	YES	YES	YES	NO

CRITERIA FOR DIAGNOSIS:

1)Discomfort (including pain at the bite site, swelling, and a local lymph node).

2)Serosanguinous seeping from the biting site

3)Enlarged, painful, draining lymph node near the bite site.

4)Discoloration: the bite site will turn a different colour.

5)Edema – The bite sites on the digits exhibit swelling(fingers, especially); regional edema appears in more than half of the bitten limb (in the absence of the Tourniquet), and the edema spreads quickly from the bitten location, for instance, within a few hours of bites on the wrist or ankle, beyond hands, feet.

COMPLETE HEMOGRAM FOR INVESTIGATIONS

Differential and Total Count:

There will be polymorph nuclear leucocytosis due to acute inflammatory reaction to snake venom.

Hemoglobin and PCV:

Initially, due to plasma extravasation, there is hemoconcentration which is followed by hemolysis or anaemia caused by bleeding.

Platelet count:

Because of the venom's direct or indirect effects of DIC, thrombocytopenia, and After a Vipera Russellei bite, EchisCarinatus. It directly correlates with how serious Russell and Saw scaled viper venomation

Peripheral Smear:

Fragmented red cells due to microangiopathic hemolysis.

Serum or Plasma:

Due to high amounts of haemoglobin and myoglobin in the blood can either be pink or brown depending on the plasma.

BIOCHEMICAL ABNORMALITIES:

Aminotransferases and muscle enzymes are increased in cases of muscle injury. Blood extravasation and hemolysis cause bilirubin levels to rise. Russell's

viper ,Hump-nosed viper bites, and Sea snake induced renal failure resulted in increase in potassium, creatinine, urea, and BUN levels .

Hyperkalemia due to rhabdomyolysis occurs by sea snake bite.Snake bite induced AKI results in metabolic acidosis.

ARTERIAL BLOOD GASES AND PH:

Respiratory and metabolic acidosis brought on by neurotoxic envenoming and infection may result in silent respiratory failure.

OXIMETRY PULSE:

The oldest non-invasive technique for tracking respiratory failure and shock

URINE ANALYSIS:

Dipsticks are used to evaluate the urine's colour and look for the presence of Blood, myoglobin, and haemoglobin.

No credible test exists to distinguish between the presence of Urine with myoglobin and haemoglobin.Microscopy can verify the presence of erythrocytes in the urine.

Red cell casts indicate glomerular haemorrhage when they are present.The first indication of an increase in capillary permeability and massive proteinuria is a sign of severe renal damage.

20 MINUTE WHOLE BLOOD CLOTTING TIME:

ADVANTAGES:

Most reliable test that can be carried out at the bed side. Doesn't need specialized training.

REQUIREMENTS:

Dry glass test tube,Syringe,Cotton,Antiseptic solution and Clean gloves.

PROCEDURE:

Take 2 millilitres of blood from the unaffected person's peripheral limb vein.Then spill the blood along the interior walls of tube test.Hold onto the test tube untouched in a vault and 20 minutes later, the test tube is gently slanted if the blood,and if the blood is still in liquid phase, the patient is said to have coagulation abnormality.

THE PROTHROMBIN TIME:

This test evaluates the common and extrinsic blood coagulation cascade pathways. It is unique to envenomation by Russell vipers, However in management it is not consistently carried out.An enzyme that activates factor X during coagulation is present, hence factor VII is not needed. The one-stage prothrombin time is the stypen time.This test carried out using the venom of the Russell's viper, which can distinguish between factor VII and factor X deficiency.

PARTIAL THROMBOPLASTIN TIME:

The process for determining the intrinsic and common pathways of coagulation but not in accordance with standard procedure for determining envenomation

Additional investigations: The electrolyte disturbances can be found via electrocardiography such as hyperkalemia and hypokalemia.

ULTRASOUND ABDOMEN:

It is used to determine the renal echoes and corticomedullary differentiation.

ANTIVENOM TREATMENT:

At the Pasteur Institute in Saigon, Albert Calmatte found the one and only unique antidote to snake venom in 1980. The venom of one or more snake species is used to immunise the horse or donkey and to prepare anti venoms.

Antivenom is made from immunoglobulin that has been separated from animal plasma.

Antivenom that has been created specifically to combat a particular snake that bit the patient should now be anticipated to neutralise the specific venom as well as the venom of related species. Antivenoms are both monovalent and polyvalent. This includes polyvalent antivenom, which is available in India. In India, equines are used to produce polyvalent antivenom against Venom of the Russell's viper, saw-scaled viper, spectacled cobra, and common krait. It can takes the shape of liquid or lyophilised powder that can be reconstituted.

DOSAGE OF ASV:

A single Russell viper injects about 63 mg of venom.6 mg of Russell's viper venom can be neutralised by one vial of ASV. Therefore, 63 mg of venom requires 10 vials to neutralise. Not all victims will need 10 vials because some may only receive injections of 63mg or less.

Additionally, they could generate pre-sensitivity in the patient. For neurotoxic/hemotoxic envenomation, it is advised to provide 8-10 vials of ASV as the first dose.As snakes inject the same ASV into adults and children, the dose is same for both.

Administration:-

ASV can be given in two different ways over the course of one hour.At a consistent rate, and the patient needs to be watched closely for 2 hours.[17](Cherian m)

Facts to keep in mind prior to/during the use of Anti Snake Venom (ASV): -

1. ASV is marketed as a liquid or solid in polyvalent form.Lyophilized medications in a 10 ml ampoule or vial.
2. Keep in mind to use and maintain the cold chain system.Users are advised to check the cold chain's integrity before usage
3. Should not initiate ASV treatment if there is no proper drugs priorly for controlling anaphylactoid reactions or anaphylaxis.
4. Test doses doesn't prevent anaphylactic shock or late serum sickness.
5. ASV is given early because it neutralises unbound venom.

6. ASV administration shouldn't be postponed or refused on the basis of a worthy case on the grounds of anaphylactic responses.

7. ASV is only required for those who exhibit clear signs and symptoms.

8. No interaction with ASV has occurred.

9. Safety status for usage in pregnancy has not been established.

INDICATIONS FOR ASV:

SYSTEMIC ENVENOMATION:

1)Hemostasis abnormalities like signs of spontaneous bleeding ,coagulopathy and thrombocytopenia symptoms.

2)Ptosis, external ophthalmoplegia, and other symptoms of neurotoxic envenoming

3)Muscle weakness and respiratory paralysis.

4) shock, hypotension, and other cardiovascular abnormalities

5)Arrhythmias, ECG alterations

6)Oliguria and elevated blood urea are signs of an acute kidney injury and symptoms of metabolic acidosis with Changes in the urine's colour

7)Muscle aches and pains are indicators of rhabdomyolysis and hemolysis and Hyperkalemia

LOCAL ENVENOMATION:

1)More than half of the bitten limb had localised edema within 48 hours

2)Rapid welling extension within a few hours of the bite with the enlargement of sensitive lymph nodes.

3) Antivenom treatment can correct the symptoms of systemic envenoming, it is possible to administer it even if the patient shows signs of systemic envenoming after several days or hemostatic abnormalities after two or more weeks.

CONTRAINDICATIONS TO ANTIVENOM:

The use of antivenom is not completely prohibited. Those having a long history of allergic response to snake venom and possibilities of developing atopic illnesses are more likely. Consequently, they should be given only in cases of systemic envenomation.

ADMINISTRATION OF INJ. ASV:

When administering vials of freeze-dried medication via the intravenous method, 10ml of sterile water is added to each vial.

Intravenous infusion or push injection:

Local site injection typically is not usually recommended.Using an intravenous push injection is a cost-effective way to administer.Slow intravenous infusion is used to administer antivenom.

Intravenous infusion: Around 10 ml of distilled water are added to the antivenom .The patient must undergo at least one hour of monitoring, so that early epinephrine can be used to diagnose and treat anaphylaxis.

ASV REACTIONS:

Acute hypersensitivity reactions: (10 mins – 3 hours)

After receiving antivenom, the patient experiences itching, usually centred on the scalp and urticaria, as well as fever, nausea, and vomiting within 10 minutes to 3 hours.abdomen pain, colic, dry cough, vomiting, and tachycardia can occur.In some instances, the patient may experience potentially fatal anaphylaxis .Hypotension, angioedema, and bronchospasm may also occurs.

They aren't actually allergic as they lack specific IgE reactivity. Those are Primarily as a result of direct or indirect complement activation by IgG aggregation and antivenom proteins that stimulate mast cell pyrogenic responses (within 1 – 2 hours) Rigors, fever, a drop in blood pressure, and vasodilation appear in the patient may result from ASV being contaminated with pyrogens during the manufacture.

Late reactions, akin to serum sickness (after 2 – 12 days):

Patient have symptoms like vomiting, diarrhoea, periarticular swellings, lymphadenopathy, fever, nausea, itching, recurrent urticaria, arthralgia, myalgia, mononeuritis , proteinuria that are due to immune complex deposition. Rarely, a patient may present with complicated nephritis and encephalopathy.

TREATMENT OF ASV REACTION:

1)Stop ASV infusion.

2)Keep IV line in place

3)Inject 0.5ml of adrenaline at a ratio of 1:1000 IM in adults and for paediatric, 0.1ml/kg body weight of 1:10,000 IM dose is given.

4)An additional 0.5ml of Adrenaline IM is administered. This might be repeated one final time however, in the great majority of cases, Adrenaline doses of two will be sufficient.

Take these additional steps: -

1. To treat bronchospasm, use a nebulizer or aerosol to provide salbutamol or terbutaline (Beta 2 agonists).
2. Administer both H1 receptor blockers when taking antihistamines.10-20 mg of chlorpheniramine maleate intravenously or intramuscularly, or promethazine 0.5–

1 mg/kg, and H2 receptor blockers like ranitidine 1 mg/kg, 0.4 mg/kg for famotidine, or 4 mg/kg slowly for cimetidine intravenously.

3. Intravenously administer corticosteroids: Hydrocortisone 2-4mg/kg or 0.1–0.4 mg/kg of dexamethasone

4. In the event of laryngeal edema, try nebulized adrenaline (5ml of 1:1000.)

5. Not to postpone intubation if a blockage of the upper airways is developing.

6. For hemodynamic instability, IV fluids should be administered.

7. Once the patient is well, the ASV can be gradually restarted after 15 to 20 minutes. Maintaining close watch of the patient. Then the drip rate should return to normal. Keep an eye on the vital signs.

LATE SERUM SICKNESS REACTION TO ASV:

Reactions to ASV that cause late serum sickness (delayed hypersensitivity) – One to two weeks after administering ASV, serum sickness may arise. An easy way to treat late serum sickness reactions is to administer prednisolone, adults take 5 mg every six hours; children receive a lower dose 0.7mg/kg/day. Additional symptomatic alleviation is provided by oral H1 antihistamines.

CARE FOR THE BITTEN PART:

Antibiotics,serratiopeptidases, analgesics, and Proper Limb Elevation should be used to treat the cellulitis. NSAIDS should not be used because may harm the kidneys. If it is large, look for compartmentalization.Consequently, fasciotomy ought to be carried out following coagulopathy treatment.

ACUTE KIDNEY INJURY IN SNAKE BITE:

Most commonly seen in Russell viper and bothropos species bite.Can happen within few hours of the bite.

DIFFERENT FUNCTIONS OF THE KIDNEYS:

The primary job of the kidneys is to remove urea, a waste product of the metabolism of amino acids. Additionally, they assist in eliminating muscle creatinine and bilirubin from haemoglobin.

The poisons produced by synthetic substances, medicines, and hormonal metabolites.Water and electrolyte balance are regulated by the glomerular filtration, tubular reabsorption, and tubular secretion.

The kidneys are crucial for preserving the acid-base balance.Kidneys excretes a small number of acids, such as sulfuric acid, phosphoric acid etc. By use of the renin-angiotensin system, it keeps the arterial pressure constant.In the process of creating erythrocytes, a hormone called erythropoietin, which encourages bone marrow to produce red blood cells,Hypoxia is the primary stimulus in production of RBC’s.

PRE-RENAL CAUSES FOR ACUTE KIDNEY INJURY:

1)Intravascular Hypovolemia from repeated vomiting, fluid Restriction in rural India as a part of treatment leads to hypovolemia, and also fluid accumulation locally in limb cellulitis leads to decreased renal perfusion leading on to pre renal AKI.

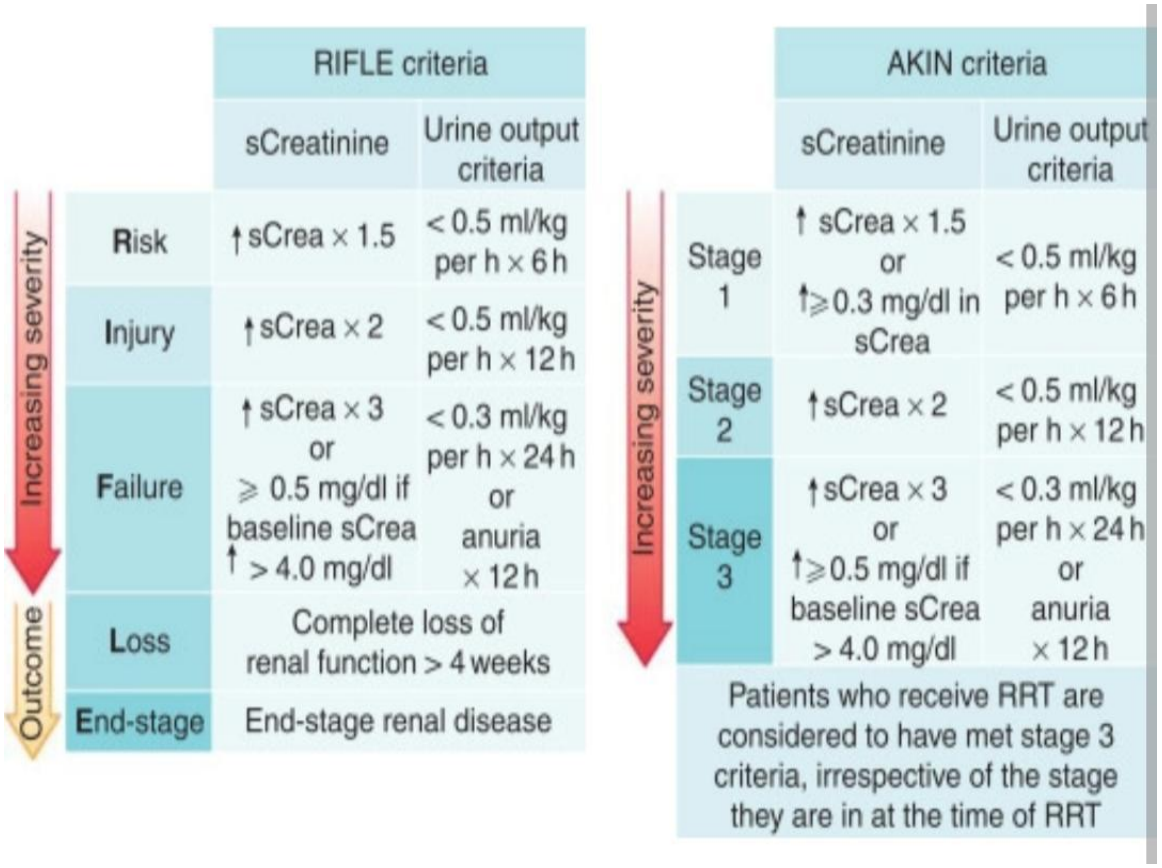
2)Increased vascular permeability is the cause of Capillary Leak Syndrome,leading onto third space volume loss and subsequent hypovolemia .Correcting hypovolemia with IV crystalloids like normal saline and Ringer's Lactate is advised. The ideal Central Venous Pressure is should be 7–10 cm of water , monitored and maintained.

3)Sometimes anaphylaxis and allergic responses to antisnake venom occur which may harm the kidneys.

CORTICAL NECROSIS:

1. Acute tubular necrosis is the most common renal lesion associated with snake bite and carries good prognosis.
2. Cortical necrosis may be diffuse or patchy.It may progress to chronic kidney disease and may need renal replacement therapy.There have been reports of papillary necrosis in Calicut. Patients with colic and hematuria may complain of passing tissues in their urine
3. Tubulo-Intestinal Nephropathy: Delayed onset (after 7 days).Biochemical testing identified non-oliguric renal failure that could be brought on by ASV or other medications used in treatment.
4. Hemorrhagic Glomeruonephritis is a fairly uncommon condition.

AKI STAGING:



		RIFLE criteria	
		sCreatinine	Urine output criteria
Increasing severity	Risk	↑sCrea × 1.5	< 0.5 ml/kg per h × 6 h
	Injury	↑sCrea × 2	< 0.5 ml/kg per h × 12 h
	Failure	↑sCrea × 3 or ⩾ 0.5 mg/dl if baseline sCrea ↑ > 4.0 mg/dl	< 0.3 ml/kg per h × 24 h or anuria × 12 h
Outcome	Loss	Complete loss of renal function > 4 weeks	
	End-stage	End-stage renal disease	

		AKIN criteria	
		sCreatinine	Urine output criteria
Increasing severity	Stage 1	↑ sCrea × 1.5 or ↑⩾0.3 mg/dl in sCrea	< 0.5 ml/kg per h × 6 h
	Stage 2	↑sCrea × 2	< 0.5 ml/kg per h × 12 h
	Stage 3	↑sCrea × 3 or ↑⩾0.5 mg/dl if baseline sCrea > 4.0 mg/dl	< 0.3 ml/kg per h × 24 h or anuria × 12 h
	Patients who receive RRT are considered to have met stage 3 criteria, irrespective of the stage they are in at the time of RRT		

MANAGEMENT OF AKI:

The majority of patients with acute renal failure are oliguric, defined as having a urine output of 20 ml or less per hour or less than 400 ml per day.For these patients, conservative treatment may be avoided, although there is a risk ,the patients in this group have an increased need for dialysis.If the patient exhibits symptoms of decreased intravascular volume, such as severe dehydration, empty neck veins, supine or postural hypotension,

Then Should go in the following manner

1)Create an intravenous access point.

2)Place a urethral catheter with strict sterility measures

3) Fluid challenge :

Administer two litres of isotonic saline over an hour ,till the central venous pressure/jugular venous pressure is normal of 8–10 cm of pressure present above the sternal angle.

While doing this, the patient should be closely watched.If pulmonary oedema appears, the fluid challenge should be halted promptly.It is fair to try an if the urine flow does not improve ,Mannitol challenge or furosamide challenge, although these are not of demonstrated value.

4)FRUSEMIDE CHALLENGE:

Challenge with furosemide 100 mg which is slowly injected (4-5 mg/minute). Give a second dose of 200 mg of furosemide if this does not cause an increase in urine output of 40 ml/hour.Try conservative management if urine output does not improve.

5) Conservative administration

If, despite these difficulties, the urine output does not improve, no additional diuretics should be administered along with fluid restriction is followed limited to the sum of the output from the previous day + “insensible loss”,which is around 500-1000 ml/day

A renal dialysis unit should be recommended for the patient.The diet should be bland, rich in calories (1700 per day), low in protein (less than 40 g/day), and high in potassium (avoid fruit and fruit juices). and are low in salt.Infections lead to tissue deterioration and elevated urea levels.They need to be avoided or treated right away with non-nephrotoxic medications.Avoid aminoglycoside antibiotics such gentamicin when using antibiotics

6). Serum potassium, urea, creatinine, and, if applicable, bicarbonate, calcium, phosphate, and pH should all be watched over frequently.

7) Management of complications like metabolic acidosis and hyperkalemia should be met and corrected to improve the general condition of the patient.

Indications for Dialysis :

1)uraemia

2) Volume overload

3)Potassium >7 mmol/l (or hyperkalaemic ECG changes)

4) Symptomatic acidosis

TREATMENT OF RENAL DAMAGE IN HEMOGLOBINURIA AND MYOGLOBINURIA:

Treating hypovolumia are metabolic acidosis forms the main stay to prevent renal damage in these conditions .Saline diuresis with sodium bicarbonate infusion is given.

Treatment of the diuretic phase of kidney damage:

This is equally fatal as the oliguric phase. Urine output increases to 5–10 litres/24 hours.The patient could develop polyuria and volume depletion, making salt intake necessary.In some people, hypokalaemia can occur.In this scenario, a potassium-rich diet (fruit and fruit juices) is advised.

Phase of renal recovery management:

After Russell's viper bite, the diuretic phase may extend for several months. In South India and Myanmar, hypopituitarism could make recovery more difficult for victims of Russell's viper bite. Hydration, corticosteroids may need to be replaced.

METHODOLOGY

This study was done at Govt. Chengalpattu Medical College hospital, during a period between April 2021 to May 2022.This is a cross sectional study which had a sample size of 150 patients

SOURCE OF DATA

The study group included 150 patients admitted to Chengalpattu Medical college hospital with history of snake bite and who satisfied inclusion and exclusion Criteria.

METHOD OF COLLECTION OF DATA

Patients were evaluated by taking a detailed history, clinical Examination and laboratory investigations. A proforma was specially Designed for data collection including all these.

INCLUSION CRITERIA

1.History of snake bite with signs of envenomation

2. Progressive elevation of serum creatinine>0.3mg/dl from baseline, a Percentage increase in the serum creatinine concentration of >50% or Oliguria of less than 0.5ml/kg/hr for more than 6hrs.

3. Age is more than 18 years.

EXCLUSION CRITERIA

1.Patients with pre existing renal Diseases with history of snake bite.

2. Extreme age groups – age more than 80 years

3.Patients with contracted kidneys with normal Renal Parameters with history of snake bite.

All patients were subjected to the following investigations

1. Hemoglobin
2. PCV
3. Platelets
4. Blood Urea, Serum creatinine,
5. USG abdomen & Pelvis
6. ECG

PROCEDURE IN DETAIL :

Patients admitted in Emergency Room, with history of Snake bite were taken into the study. Some of the patients were initially treated in some hospitals and referred to our hospital with acute Kidney injury was also taken. Details regarding initial treatment, renal parameters have been collected.

Acute kidney injury was defined according to AKIN criteria. Patients were classified into 4 groups- NO AKI, AKIN 1, AKIN 2, AKIN 3 by monitoring urine output and Serum Creatinine. CBC- Hb, PCV, and Platelet count was monitored daily. Renal parameters were monitored daily.

STATISICAL ANALYSIS

The study design was a cross sectional study. All data collected were noted using a structured proforma, including the investigations. Data was analysed using statistical package And SPSS structured software to find out the proportion of acute kidney Injury among 150 patients, and their clinical profile and outcome of them.

ETHICAL CONCERNS: As per the institution protocol.

CONSENT: Informed consent was taken as per standard procedure that is followed in the institution.

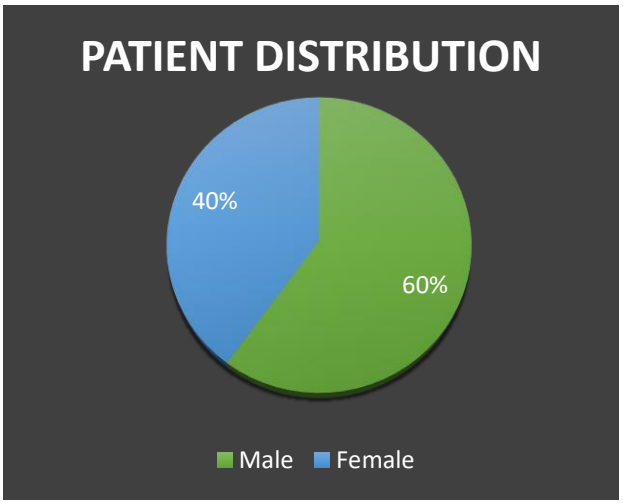
RESULTS AND OBSERVATIONS

The study was cross sectional study and 150patients were selected who fulfils the criteria for the study. Following parameters were observed in our study.

GENDER WISE DISTRIBUTION OF PATIENTS

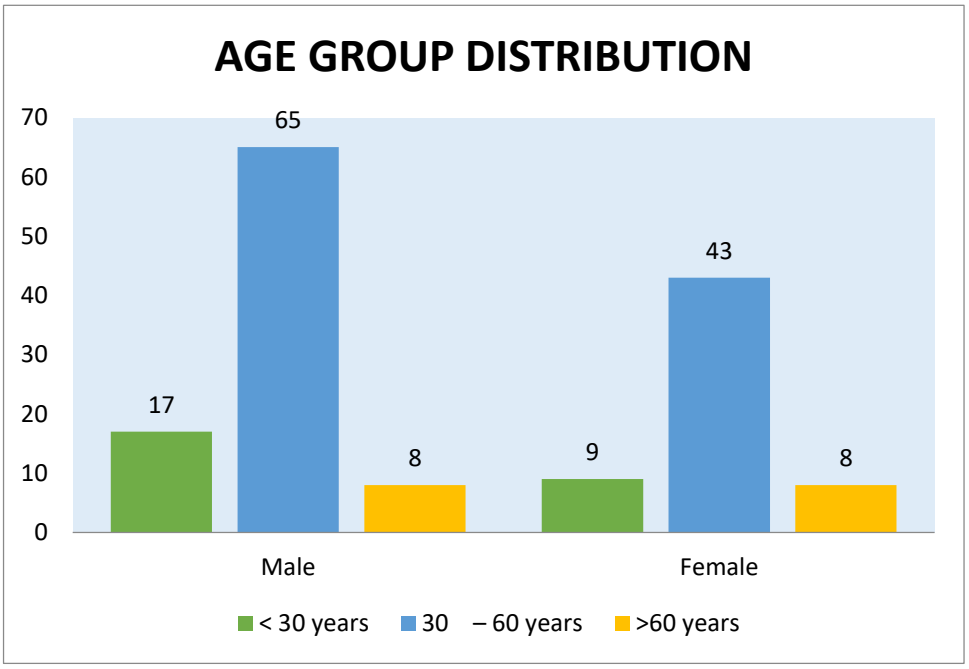
GENDER	No. of Patients	Percentage
MALE	90	60
FEMALE	60	40

In total number of patients observed in our study is 150. Among them 90 patients are male and 60 patients are female.



AGE GROUP DISTRIBUTION WITH GENDER

Gender	< 30 years	30 – 60 years	>60 years	Chi square	P value
Male	17	65	8	0.982 Df=2	0.612
Female	9	43	8		
Total	26	108	16		

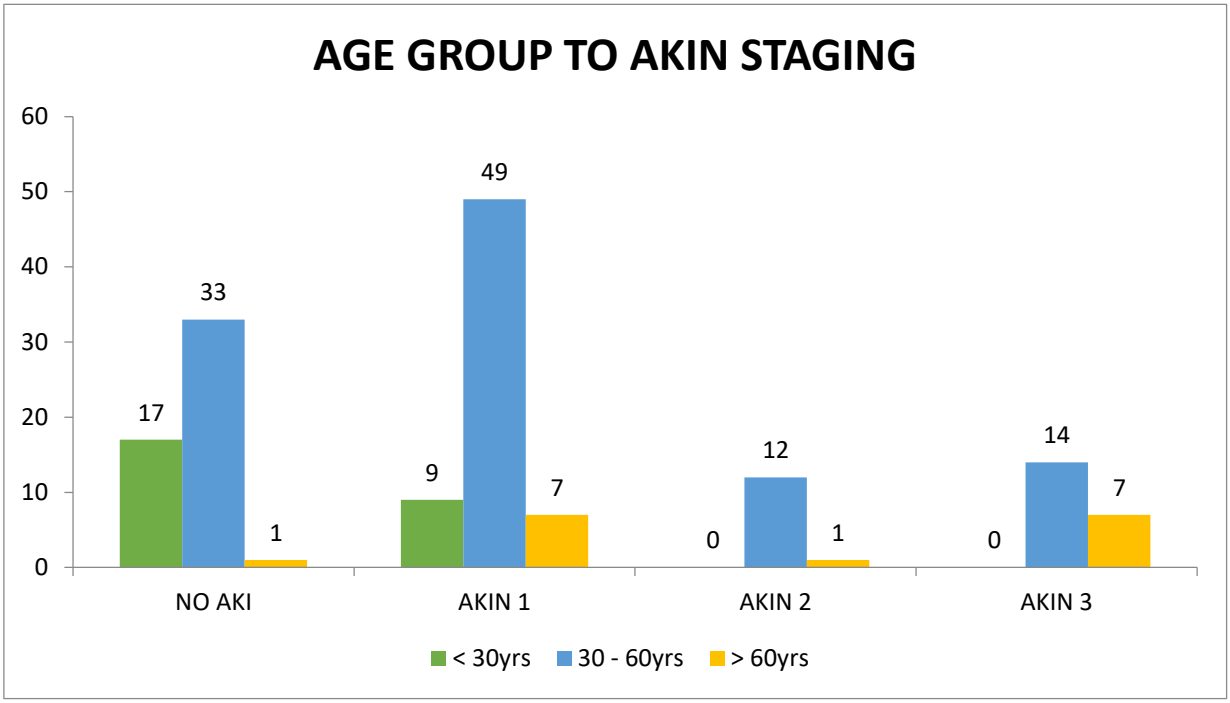


AGE GROUP DISTRIBUTION WITH GENDER

Most of the patient in both sex groups are in between 30-60 years, only Few patients are in

more than 60 years of age. Most of the patients do not have any co-morbidities. Mean age is 41

years with minimum age of 18 years and maximum age of 80 years



AGE GROUP TO AKIN STAGING

		AKIN				Chi square	P value
		NO AKI	AKIN 1	AKIN 2	AKIN 3		
Age_grp	< 30yrs	17	9	0	0	29.037 Df=6	<0.001*
	30 - 60yrs	33	49	12	14		
	> 60yrs	1	7	1	7		

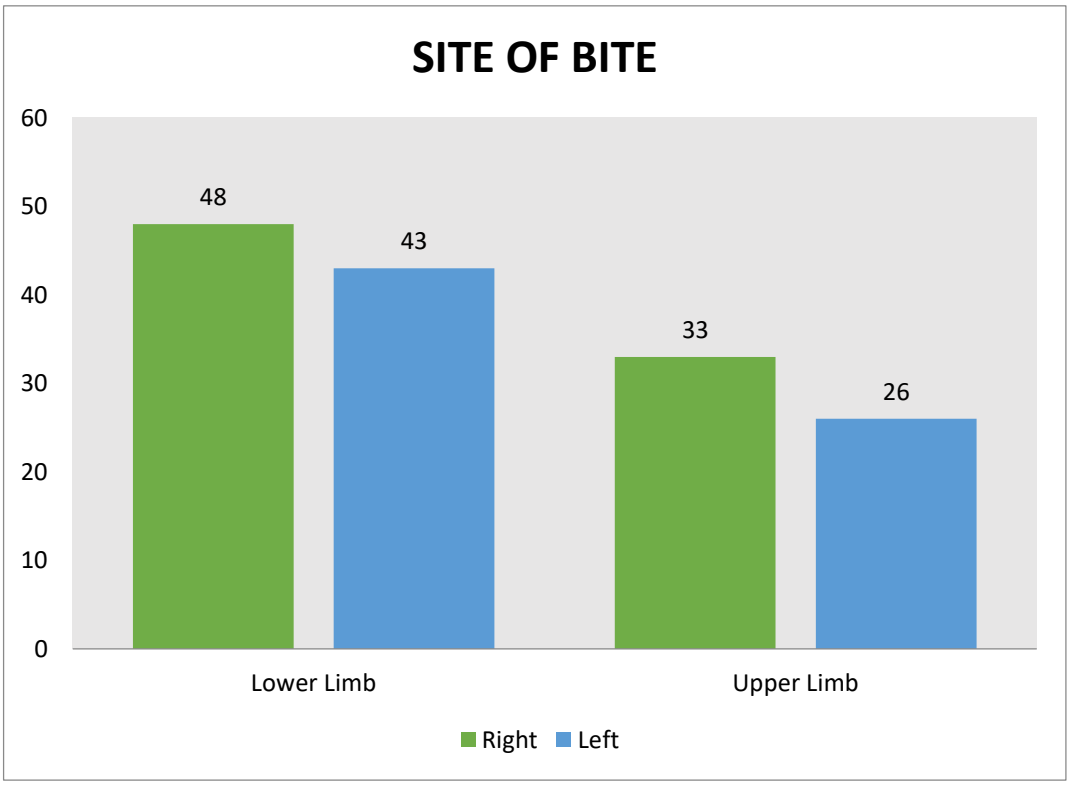
*means –significant

In more than 60 years age group 15 out of 16 persons had AKI

Whereas in middle age group, 75 out of 108 people had AKI(70%)

SITE OF BITE

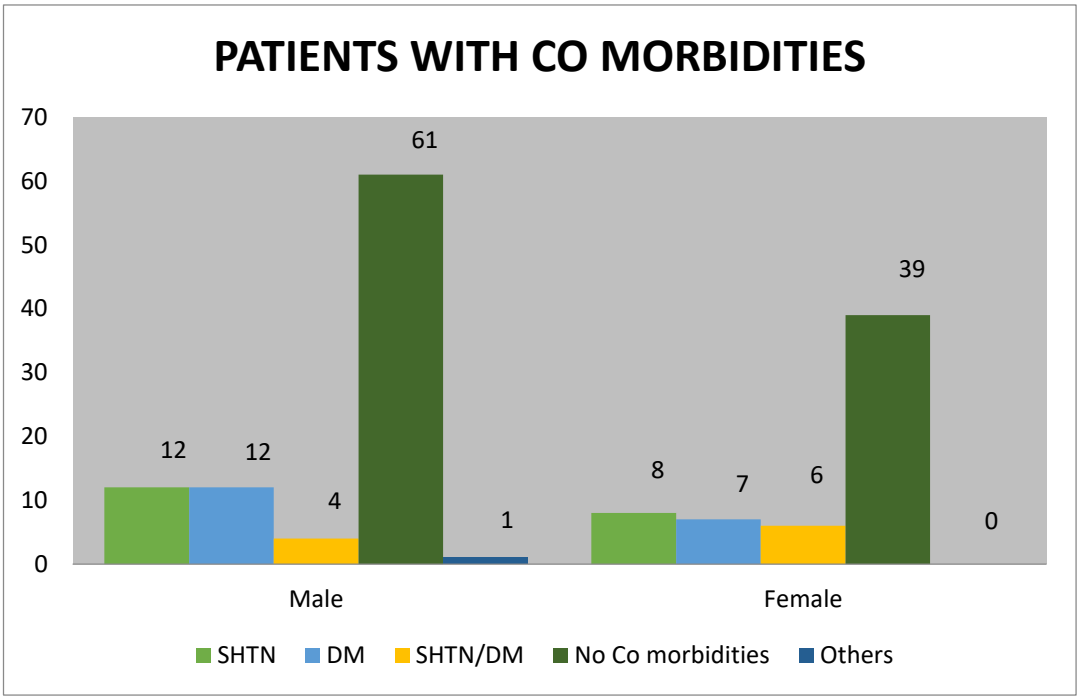
Site of bite	Right	Left	Total
Lower Limb	48	43	91
Upper Limb	33	26	59



Right lower limb is the commonest site of bite . Bite In the hand occurred in patients working in the field particularly agricultural workers.Some of the patients had been bitten over shoulder, thigh and one patient had been bitten over the face. As most of them are field workers, bare foot walkers, dorsum of foot and ankle were the most common areas of bite.

PATIENTS WITH CO MORBIDITIES

Gender	SHTN	DM	SHTN/DM	Without Co morbidities	Others	Chi square	P value
Male	12	12	4	61	1	2.454 Df=4	0.653
Female	8	7	6	39	0		
Total	20	19	10	100	1		



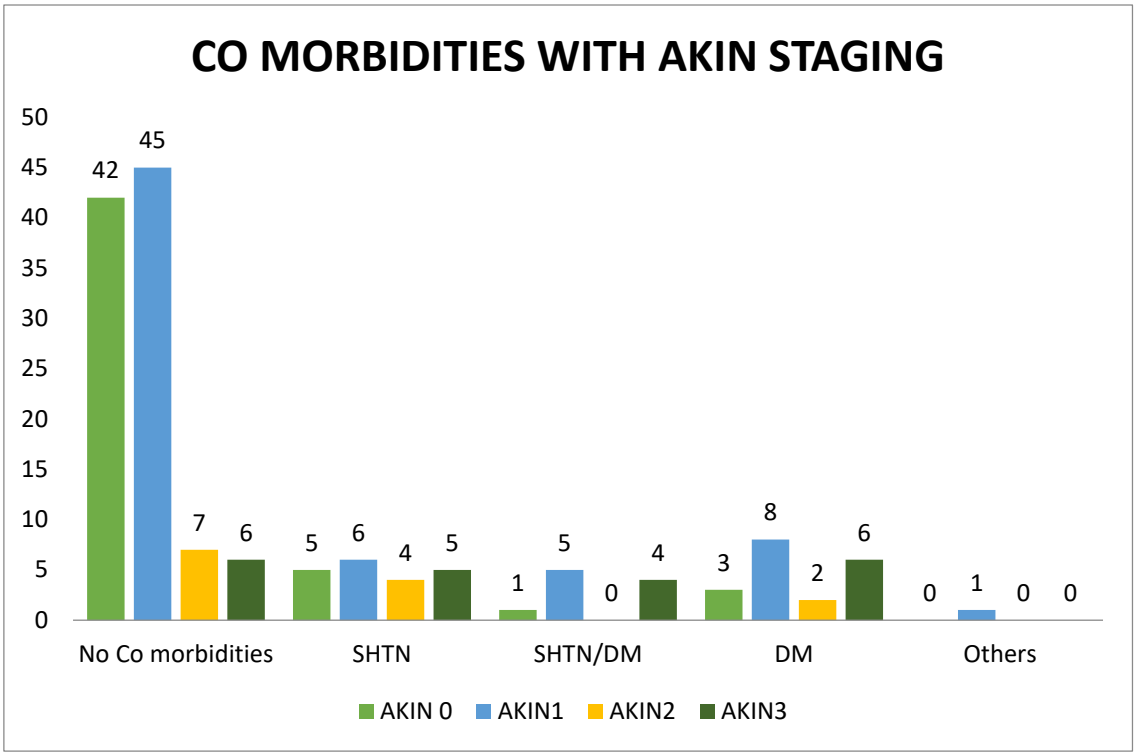
Most of the patients in our study do not have any co-morbidities.

66% of patients (100 cases)in our study were not having any co-morbidities.

One patients was HIV positive, on ART.

10 patient was both hypertensive and diabetic.

20 patients are hypertensives on regular anti-hypertensive drugs and hypertension was under control



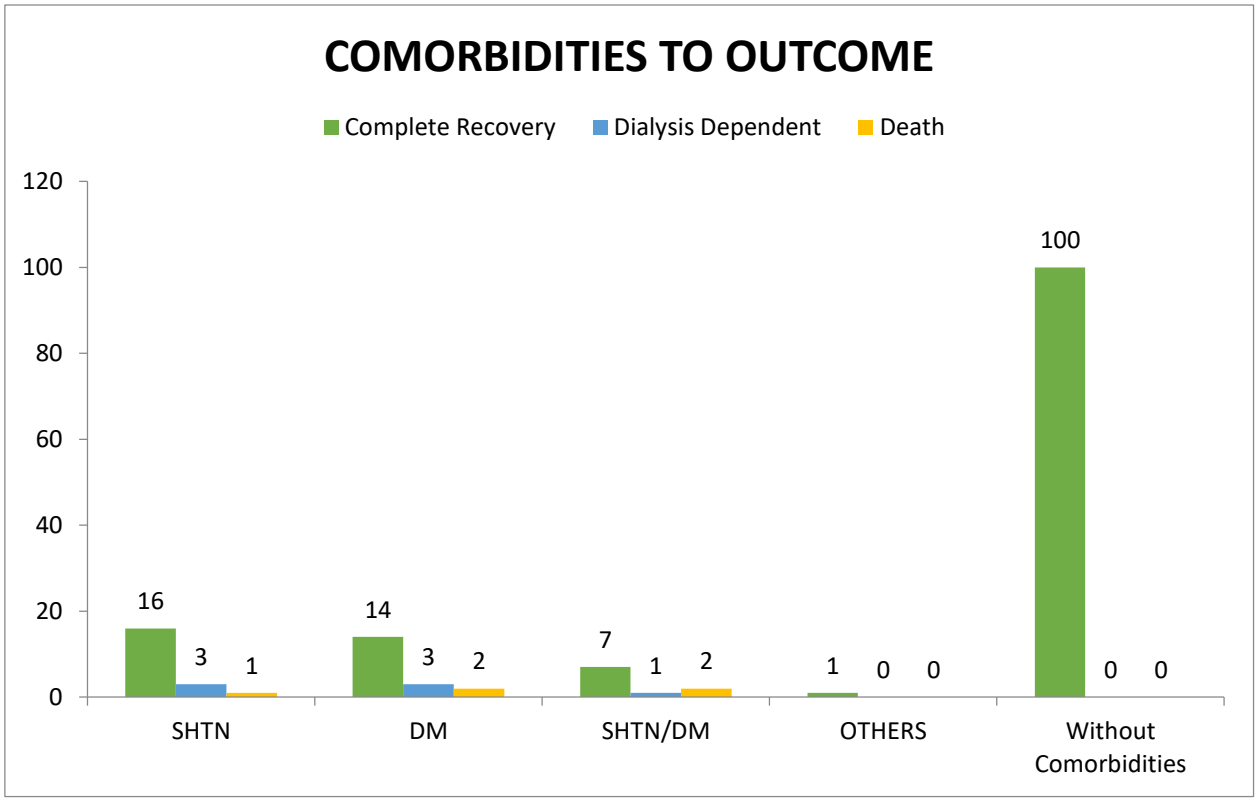
CO MORBIDITIES OF PATIENTS RELATED TO STAGING OF AKI :

Co morbidities	NO AKI	AKIN1	AKIN2	AKIN3	TOTAL	Chi square	P value
Without comorbidities	42	45	7	6	100	27.757 Df=12	0.006*
SHTN	5	6	4	5	20		
SHTN/DM	1	5	0	4	10		
DM	3	8	2	6	19		
Others	0	1	0	0	1		

*- means significant.

Among 100 patients without comorbidities 58 patients developed AKI (58%)

Whereas patients who had comorbidities 41 out of 50 patients developed AKI (82%).



COMORBIDITIES TO OUTCOME

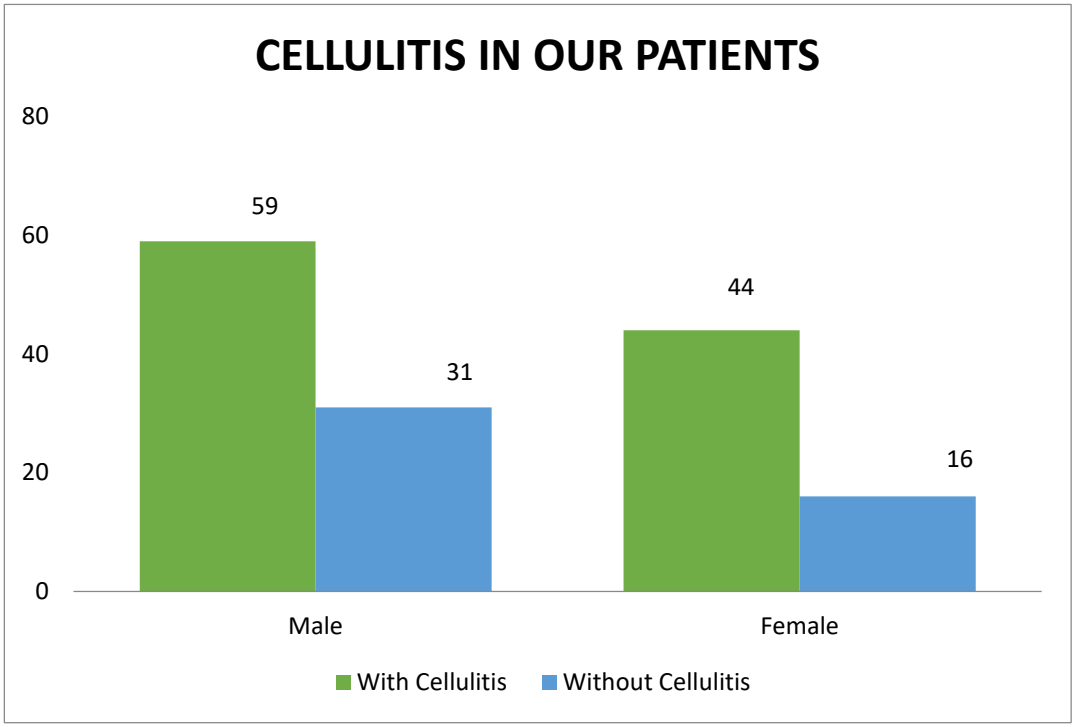
		Outcome			Chi square	P value
		Complete Recovery	Dialysis Dependent	Death		
Comorbidities	SHTN	16	3	1	31.986 Df=8	<0.001*
	DM	14	3	2		
	SHTN/DM	7	1	2		
	OTHERS	1	0	0		
	Without Comorbidities	100	0	0		

*means –significant

In our study all patients who had no comorbidities recovered completely from AKI. However among 50 patients with comorbidities 38 had complete recovery (76%).

CELLULITIS IN OUR PATIENTS

Gender	Cellulitis	Without Cellulitis	Chi square	P value
Male	59	31	1.012 Df=1	0.314
Female	44	16		
Total	103	47		



Cellulitis is more common with male, corresponds to 62 % in total Cellulitis. In my study 90 male patients were admitted, among them 59 (65%) patients developed cellulitis.

In female patients 44/60 which is 73%development of cellulitis is depend upon the species of snake bites, and potency of the venom and not depends upon the limbs. But Progression of cellulitis is depends upon the effective treatment given.

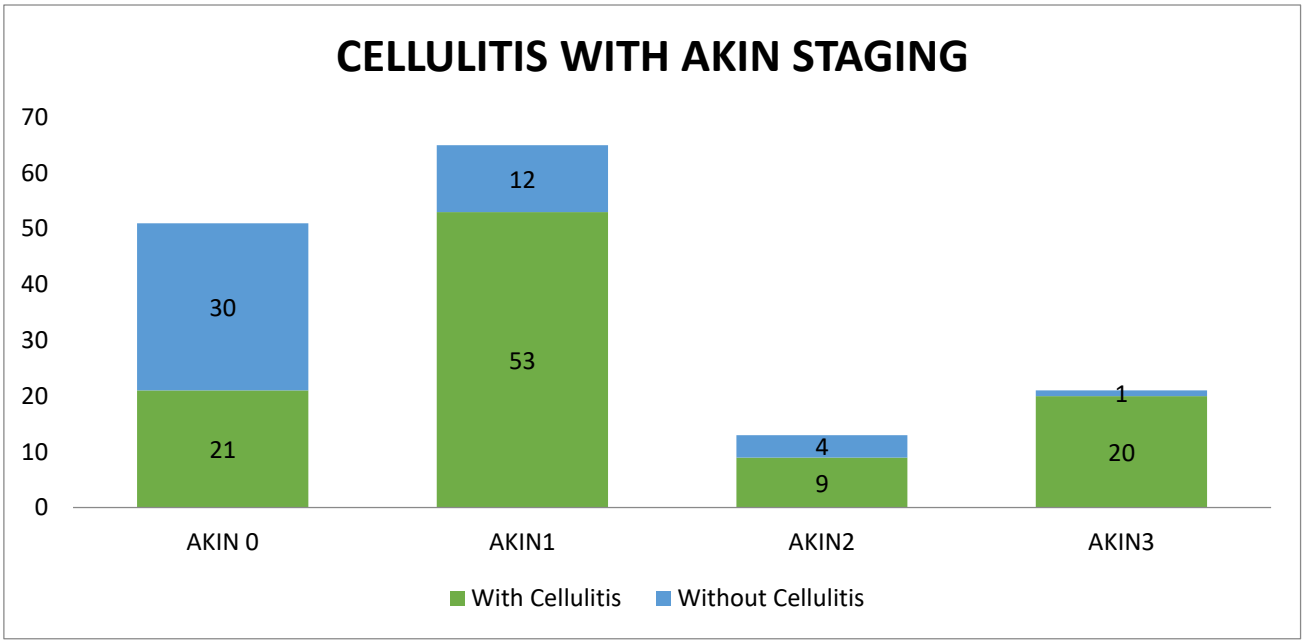
CELLULITIS IN PATIENTS RELATED TO STAGING OF AKI

Cellulitis	NO AKI	AKIN1	AKIN2	AKIN3	Total	Chi square	P value
With Cellulitis	21	53	9	20	103	29.812 Df=3	<0.001*
Without Cellulitis	30	12	4	1	47		

*- means significant

Among 51 patients in no AKI group, 21 patients had cellulitis And 30 patients didn't have cellulitis.

20 patients in akin3 group, Only one patient didn't have cellulitis. This association is significant as p value is <0.001. AKI can be expected in patients with severe cellulitis. Accumulation of litres of fluid into the bitten limb as cellulitis may be one of the reason for pre-renal AKI in this group.



THROMBOCYTOPENIA WITH AKIN STAGING

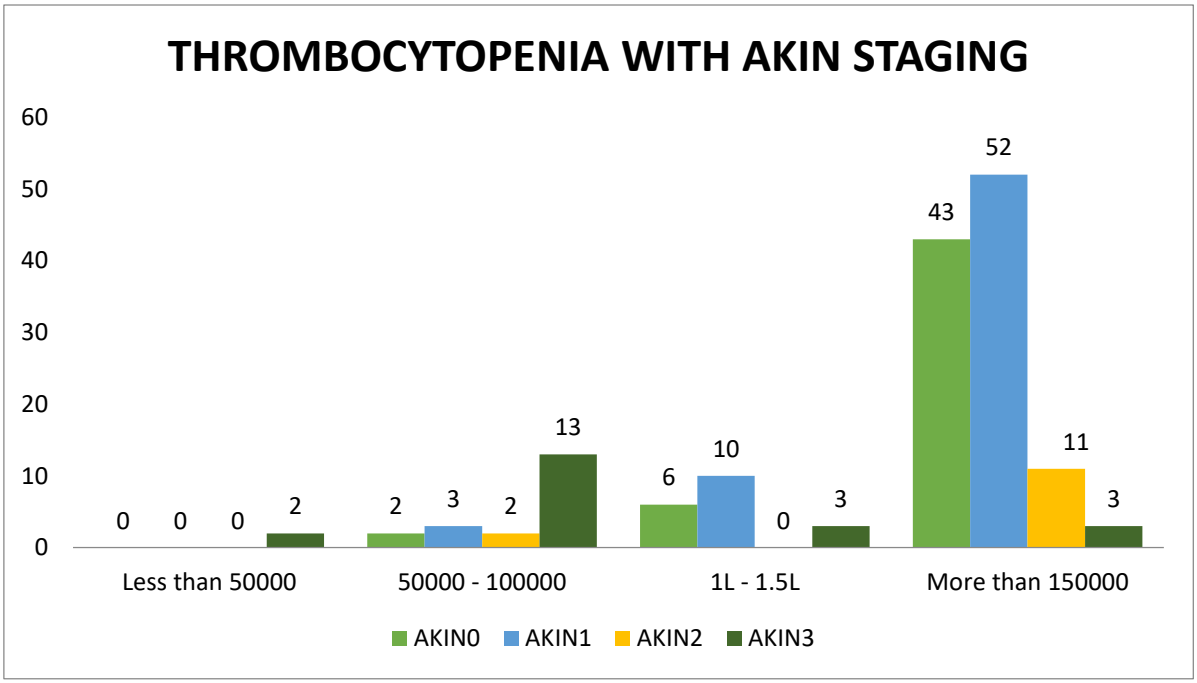
PLC	NO AKI	AKIN1	AKIN2	AKIN3	Chi square	P value
<0.5L	0	0	0	2	70.218 Df=9	<0.001*
0.5 – 1L	2	3	2	13		
1 – 1.5L	6	10	0	3		
>1.5L	43	52	11	3		

*- means significant

Thrombocytopenia was present in most of the patients with AKI. In AKIN 0 group, only 6 patients had mild thrombocytopenia in the range of 1 – 1.5 Lakh/mm3.

In AKIN 3 group,18/21 patients (84%) had thrombocytopenia, among them 10% of patients had very severe thrombocytopenia..

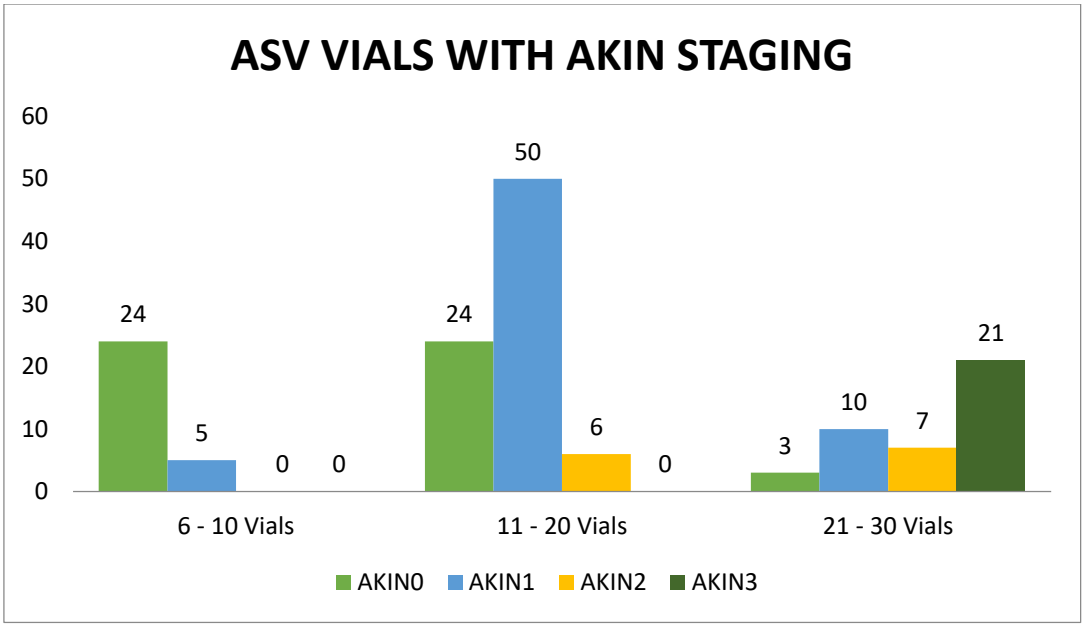
Hence thrombocytopenia canbetaken as a marker to predict severity of AKI. Also thrombocytopenia is directly proportional to severity of AKI in our study.



AKIN STAGING IN RELATION TO NUMBER OF VIALS REQUIRED TO NEUTRALISE VENOM (CLINICALLY)

ASV Vials	NO AKI	AKIN1	AKIN2	AKIN3	TOTAL	Chi square	P value
6-10	24	5	0	0	29	105.785 Df=6	<0.001*
11-20	24	50	6	0	80		
21-30	3	10	7	21	41		

*- means significant



In patient who required less than 10 vials group, only 5 people(17%) developed AKI.

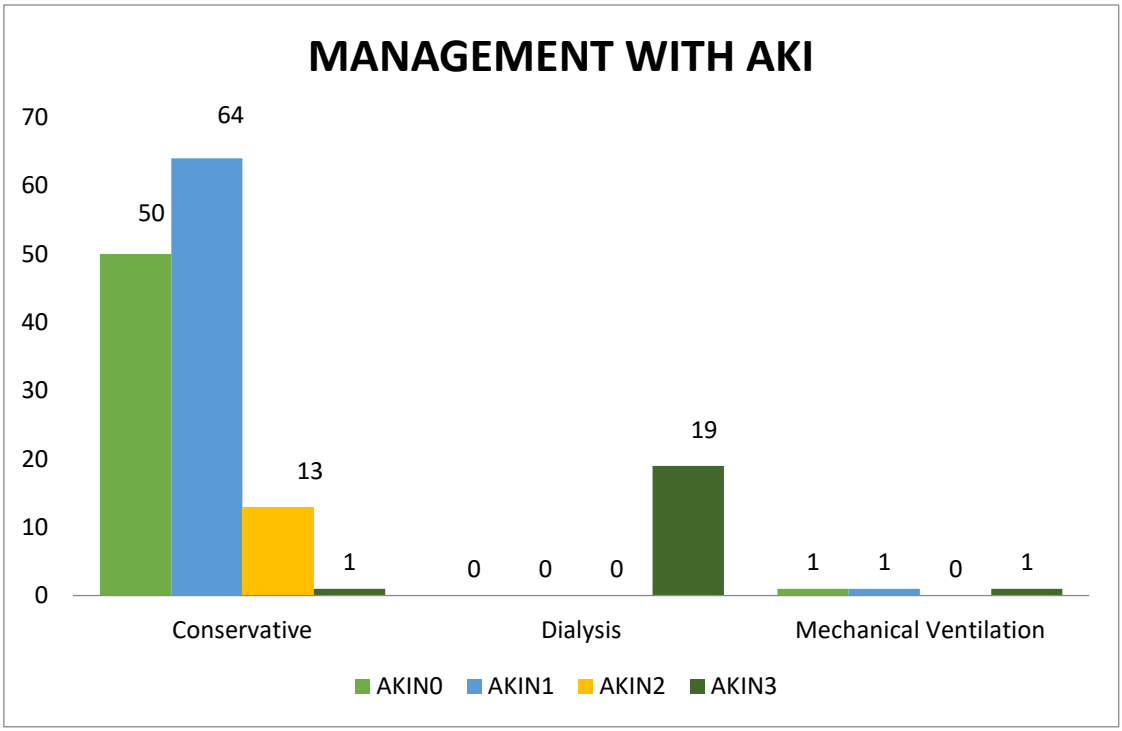
Patient who required 11-20 vials of ASV , 24 patients didn't develop AKI and 56 patients developed AKI. It is a sign of moderate to severe envenomation.

In third group, Patient who required high amount of ASV about 21-30 vials , 21 patients among 41 developed severe AKI. Maximum ASV vials given in these patient was 30 vials.

MANAGEMENT RELATED TO AKIN STAGING

Management	**NO AKI**	**AKIN1**	**AKIN2**	**AKIN3**	**TOTAL**	**Chi square**	**P value**
Conservative	50	64	13	1	128	134.426 Df=6	<0.001*
Dialysis	0	0	0	19 (13- HD 6-PD)	19		
Mechanical Ventilation	1	1	0	1	3		
Total	51	65	13	21	150		

*- means significant.HD - Hemodialysis, PD – Peritoneal dialysis.

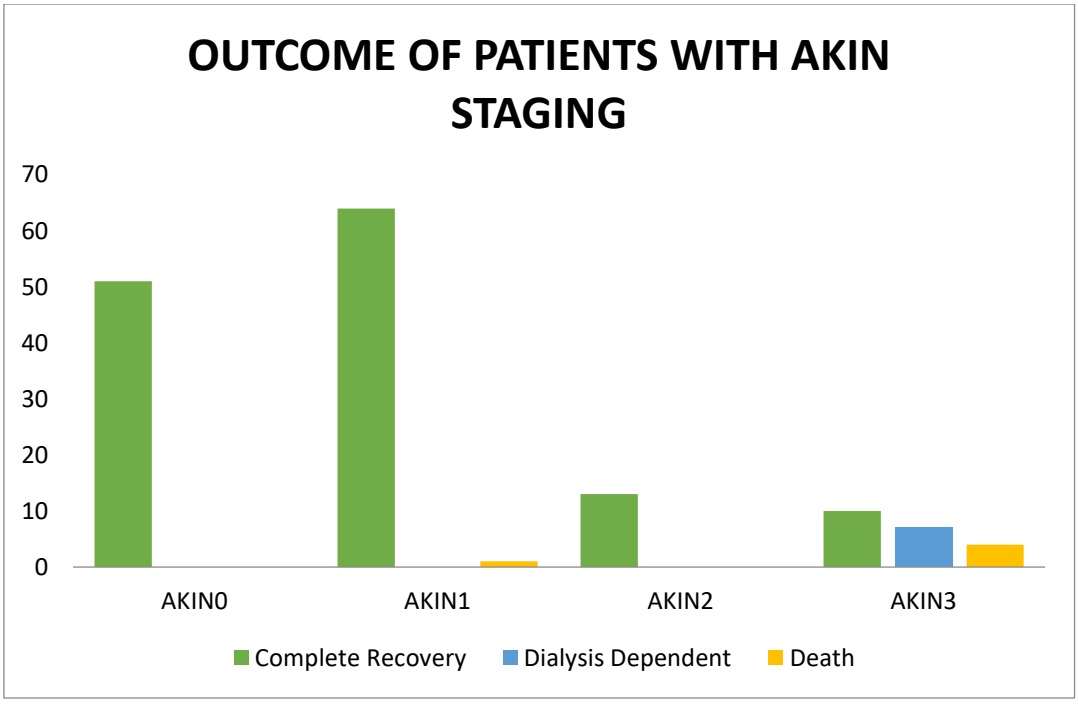


In our study among 150 patients, 99 patients developed acute kidney Injury. Among them 65 patients are in AKIN 1 stage and 1 death occured and other patients recovered completely without in need of dialysis. 13 Patients developed AKIN stage 2, among them no one required dialysis and all of them recovered completely . 21 patients developed AKIN stage 3. Among them 19 patients required dialysis and 4 patients died .

OUTCOME OF PATIENTS WITH AKIN STAGING

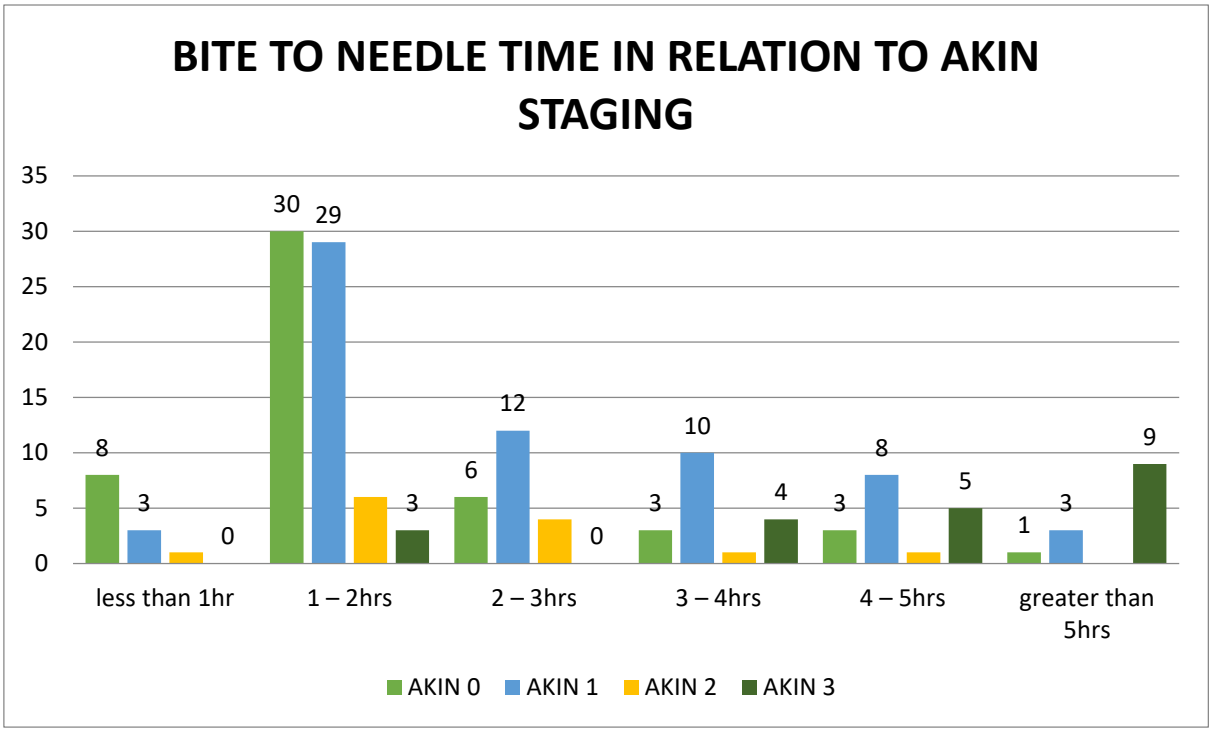
Outcome	NO AKI	AKIN1	AKIN2	AKIN3	Total	Chi square	P value
Complete Recovery	51	64	13	10	138	66.555 Df=6	<0.001*
Dialysis Dependent	0	0	0	7	7		
Death	0	1	0	4	5		

*- means significant



In our study, 99 patients developed AKI. 92% of patients recovered completely. 64 patients with AKIN 1 ; 13 patients with AKIN2 and 1 patients with AKIN 3 recovered with conservative management.

7 patients become dialysis dependent for more than a month and progressed to chronic kidney disease and advised to monitor renal parameters regularly and 5 patient died in hospital, 4 of them in stage3 AKIN and one person in stage 1 AKIN.



BITE TO NEEDLE TIME IN RELATION TO AKIN STAGING

AKIN Staging	Bite to needle time												Total	Chi square	P value
	<1hr		1 -2hrs		2 -3hrs		3 -4hrs		4 -5hrs		>5hrs				
	N	%	N	%	N	%	N	%	N	%	N	%			
NO AKI	8	15.7	30	58.8	6	11.8	3	5.9	3	5.9	1	2.0	51	60.248	<0.001*
AKIN 1	3	4.6	29	44.6	12	18.5	10	15.4	8	12.3	3	4.6	65	Df=15	
AKIN 2	1	7.7	6	46.2	4	30.8	1	7.7	1	7.7	0	0	13		
AKIN 3	0	0	3	14.3	0	0	4	19.0	5	23.8	9	42.9	21		
Total	12	8	68	45.3	22	14.7	18	12.0	17	11.3	13	8.7	150		

*- means significant

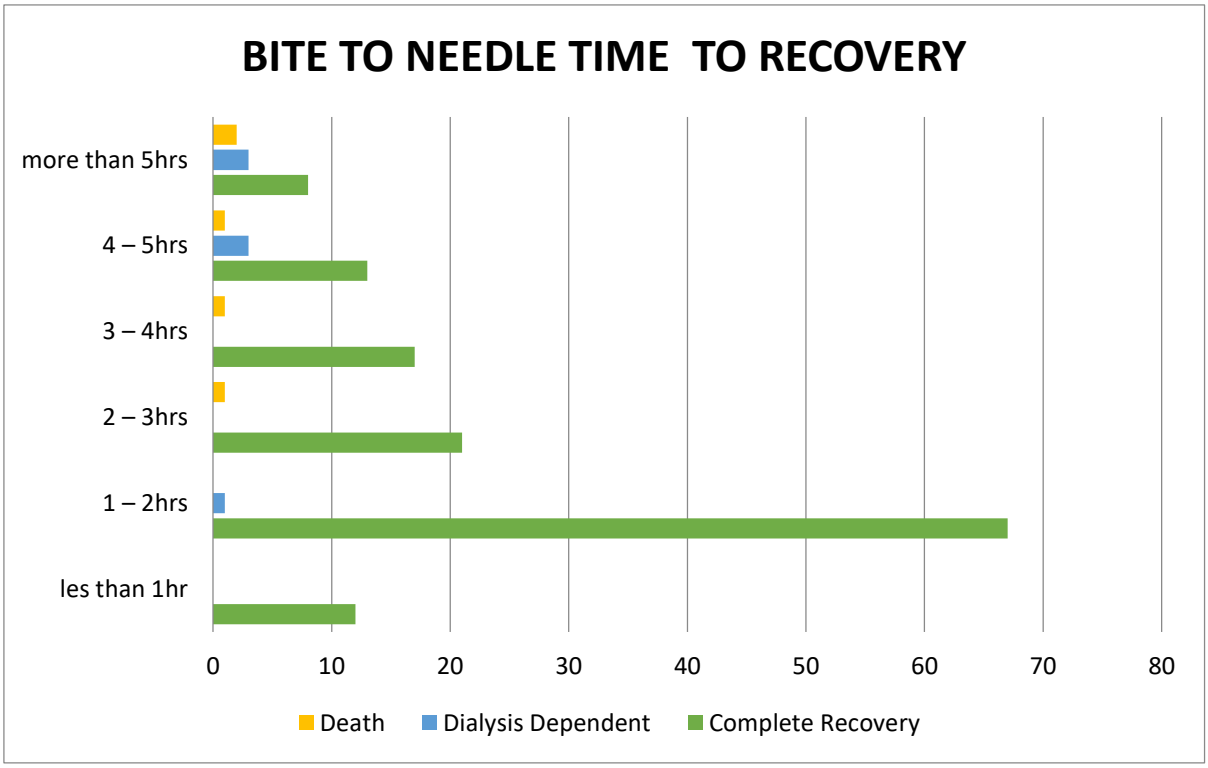
In patients without acute kidney injury 98% of patients had a BNT of <5 hours.

On the other hand In AKIN 3 group 43% patients had a BNT>5 hours.

BITE TO NEEDLE TIME TO RECOVERY

Bite to needle time	OUTCOME			Chi square	P value
	Complete Recovery	Dialysis Dependent	Death		
<1hr	12	0	0	30.716	<0.001*
1 – 2hrs	67	1	0		
2 – 3hrs	21	0	1	Df=10	
3 – 4hrs	17	0	1		
4 – 5hrs	13	3	1		
>5hrs	8	3	2		
Total	138	7	5		

*- means significant



In our study, within 1 hour of bite to needle time ,all the 12 patients recovered completely .

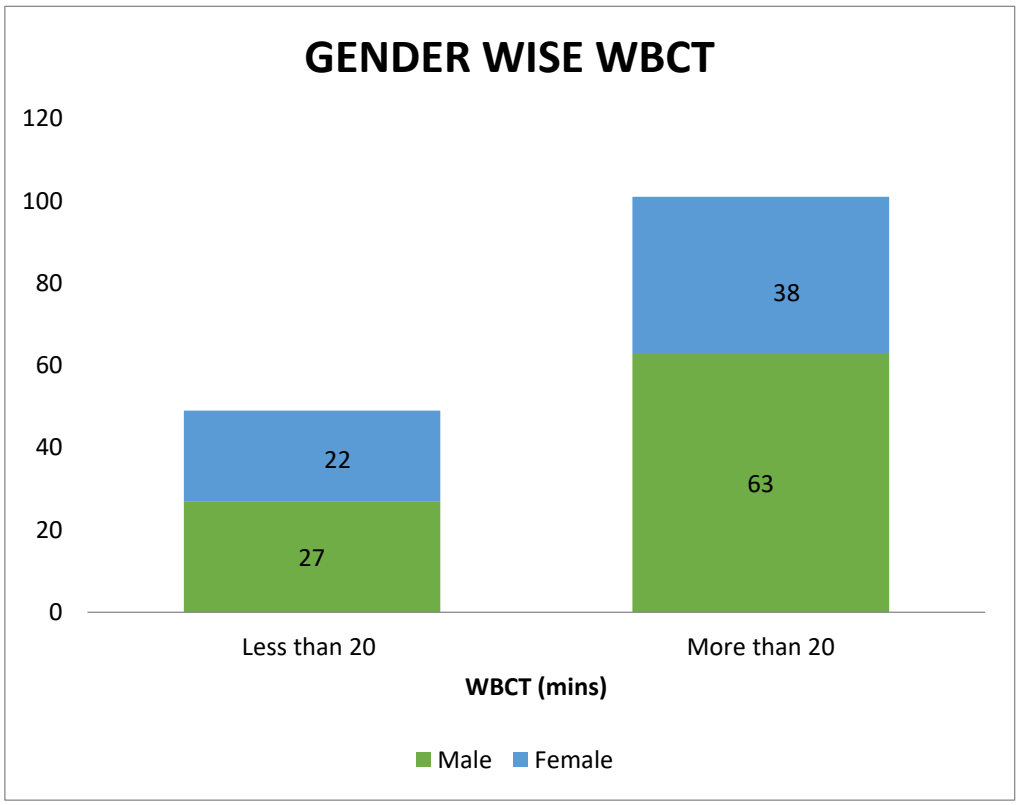
In 1 -2 hrs group ,one patient needed dialysis and 67 patients recovered completely.

After 4 hours, 6 patients needed dialysis among 30 patients and 21 patients recovered completely .

Total deaths were 5 .Among them 2 patients had bite to needle time more than 5 hours.

GENDER IN RELATION TO WHOLE BLOOD CLOTTING TIME

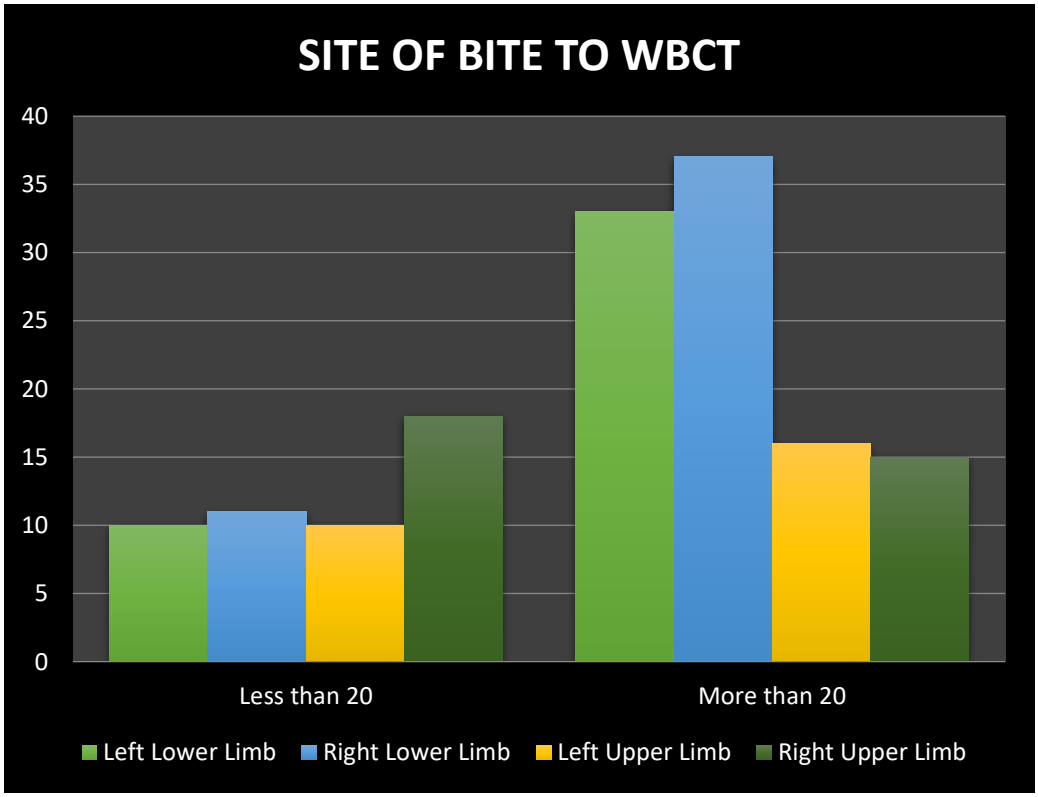
GENDER	WBCT		Chi square	P value
	<20	>20		
Male	27	63	0.727 Df=1	0.394
Female	22	38		
Total	49	101		



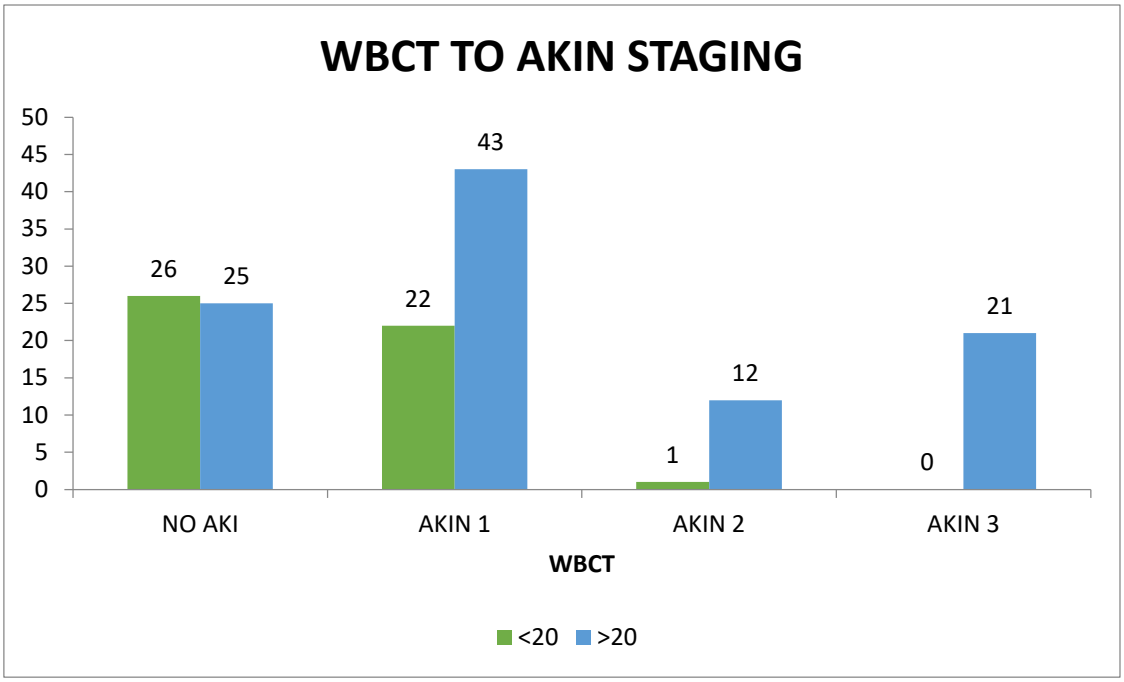
SITE OF BITE IN RELATION TO WHOLE BLOOD CLOTTING TIME

Site of bite	WBCT		Chi square	P value
	<20	>20		
Left Lower Limb	10	33	11.384 Df=3	0.01*
Right Lower Limb	11	37		
Left Upper Limb	10	16		
Right Upper Limb	18	15		

*- means significant



In our study ,people who had a bite on lower limb, had WBCT more than 20 minutes (70%)



WHOLE BLOOD CLOTTING TIME TO AKIN STAGING

WBCT	NO AKI	AKIN 1	AKIN 2	AKIN 3	Chi square	P value
<20	26	22	1	0	21.692 Df =3	<0.001*
>20	25	43	12	21		

*means –significant

In WBCT<20 min group 49% patients developed AKI

Whereas in>20 min group, 76% patients developed AKI.

DISCUSSION

In our study, 150 patients admitted with poisonous snake bites with features of envenomation. Various parameters were observed and stratified into groups and followed up till discharge.

GENDER VARIATION WITH SNAKE BITE :

In our study, 90 patients were male and 60 patients were female. Male population is more than female since most of the outdoor activities, farming, field works are done by Males usually. In 1992, 14Hati AK et al, have done one epidemiological survey snake bite in the district of the Burdwan, West Bengal. In that study 54.27% were male patients and 45.23% were female patients.

AGE GROUP DISTRIBUTION :

Most of the patients are in 30 to 60 years of age. Maximum age was taken into my study was 80 years. Most of the patients were young adult Patients, active farmers in rural Tamilnadu. To avoid age related changes In the Kidney (GFR changes) extreme age group population was not taken into the study .In 2006 to 2008, Chattopadhyay et al did a study about snake bite in same district mentioned above where snake bites are more common. In that study,Patients in the age group of 21-40 years contributed to nearly 60 % of total population and among them 60.47% are male patients.

SNAKE IDENTIFICATION :

Most of the patients (60%) have not identified the snake. Few victims brought the snake to the hospital and most of the patients were not able to differentiate Viperpatients admitted with history of cobra bite with typical features and required

ventilator and recovered completely . Most of the patients had signs and symptoms of Russell Viper bite envenomation.

COMMON SITES OF SNAKE BITE :

In our study, snake bites most frequently occur on the lower limbs, particularly the right lower limb. The majority of them are agricultural workers who were accustomed to working barefoot and through the meadows. Thus, the dorsum of the foot is most frequently bitten, followed by the lower third of the leg. These patients were frequently bitten over the dorsum of their hands. A few patients, had bites over their thighs, backs, shoulders, and face.

INCIDENCE OF ACUTE KIDNEY INJURY :

In our study among 150 randomly selected snake bite patients with features of Systemic envenomation, 99 patients developed acute kidney Injury with an incidence of 66%.

CO-MORBIDITIES IN PATIENTS:

Among 50 patients, 20 patients had hypertension, 19 patients had diabetes, 10 patients had hypertension and diabetes, one patient was HIV positive on Pre-ART.

Other co morbidities were not taken into the study. Among 50 patients, 21 people developed severe Acute Kidney Injury (AKIN2, AKIN3). 14 patients required dialysis and 2 patients become dialysis dependant, because development of patchy cortical necrosis, which was proven by renal biopsy.

4 deaths were occurred in these group. Maulita P Kapadia et al conducted one study regarding acute kidney injury of all causes with co-morbidities. In that study, more than 50% of patients with co-morbidities developed AKI.

CELLULITIS IN RELATION TO DEVELOPMENT OF ACUTE KIDNEY INJURY:

In our study 90 male patients were admitted, among them 65% patients developed cellulitis, and 73% female patients developed cellulitis.

Cellulitis development is dependent on the type of snake that bit you, the venom's potency, and not the affected limbs. However, the effectiveness of the treatment relies on how quickly the infection spreads.In individuals with severe cellulitis, AKI is a possibility because of Collection of litres of fluid into the biting limb.

Cellulitis that progresses quickly is one sign that the initial treatment should be repeated.Secondary bacterial infection, tissue necrosis,vascular occlusion and compartmental syndrome make the development of AKI due to the release of inflammatory substances which damages the Kidney vascular endothelium .

Athappan et al : "Regional lymphadenopathy was another significant Independent factor for ARF. Just as cellulitis, Regional lymphadenopathy can be a bedside indicator of the amount of toxin released by the snake bite"[18](athappan et al)

CELLULITIS AND LYMPHADENOPATHY RELATED TO ACUTE KIDNEY INJURY:

Both of them are signs of severe local envenomation, both was present in 50 male patients and 36 female patients. Hence most of the patients are Having both cellulitis and lymphadenopathy in our study[19](chetan). It is a sign of Viper bite also. 21 /86 (25%) of patients in these group developed stage 3 AKI, and more than 80% required dialysis and 7 patients become dialysis dependant and 4 patient died of AKI complications.

In 2013, Mrudul V Dharod et al done a study and concluded that "92% patients with AKI had moderate to severe cellulitis. On the other hand, only about 55% patients without AKI had moderate to severe cellulitis".Rapidly progressive cellulitis with lymphadenopathy is one of the early predicting sign of AKI and outcome. It is directly proportional to amount and potency of venom.

PROLONGED COAGULOPATHY WITH ACUTE KIDNEY INJURY:

This strategy of initiating Inj.ASV and monitoring alongside local envenomation indications is used in our institution. Few individuals in our study exhibited merely local indications of envenomation, and more than 80% of patients had prolonged clotting times.

The reversal of coagulation problems in nearly 20% of cases required blood products like FFP and whole Blood in addition to In.ASV. A few individuals experienced hematuria, one had hematemesis.. Patients with normal whole blood clotting time did not suffer acute kidney damage.Thus, it could be considered a risk factor for AKI. "101 (59.06%) snakebite patients were presented with 20 min WBCT

> 20 min and of which 55 (54.45%) patients were suffering from AKI" - (Jayanta Paul et al).

BITE TO NEEDLE TIME AND DEVELOPMENT OF ACUTE KIDNEY INJURY :

137 patients were admitted in hospital before 5 hours, among them 50 patients did not develop AKI, 62 patients developed AKIN 1 and 13 patients developed AKIN 2 and 12 patients developed AKIN 3[20]

Among patients admitted before 5 hours, 12patients admittedimmediately within one hour, among them 8 didn't develop AKI, and no-one developed AKIN 3. In more than 5 hours group, 1 people didn't develop AKI, but 9 peoples developed akin3.

In 2012, Jayantapaul[21] et al have done small study in Gujarat, Mean time in between snakebite and administration of ASV (minutes) 110.22 ± 7.60 was observed and mean patients developed acute kidney injury is 66.39 ± 4.3659

Suchithra N et al[22]. stated in their study of Snakebite envenoming in Kerala, South India; " Those who received ASV early (bite to needle time < 6hrs) had more severe local envenoming than those who received ASV late (bite to needle time > 6 hrs), but latter group were more likely to suffer complication and those who received ASV late had a higher risk of developing acute renal failure"

Sharma et al.stated that median bite to hospital time was 9 hrs and delayed In admission was more prone to develop acute kidney injury.

Narvencar K et al [23]found that Correlation between early administration of Anti snake venom was beneficial in preventing development of acute Kidney injury, however severe was the systemic envenomation

Early administration of antivenom has been demonstrated to completely reverse all clinical manifestations of snake envenomation. But some studies stated that the early administration of Inj.ASV cannot be too strongly emphasized to prevent development of AKI in snakebite Patients." – UM.Natarajan et al , SJAMS42

So early admission sometimes may not prevent development of AKI. But delay in admission is definitely resulted in bad prognosis, sometimes death may occur in those patients.

THROMBOCYTOPENIA WITH AKIN STAGING AND OUTCOME:

Thrombocytopenia was present in almost all patients with AKI. In AKIN 0 group, only 8 patients had mild thrombocytopenia and in AKIN 3 group, 18/21 patients (85%) had thrombocytopenia, among them 10% of patients had very severe thrombocytopenia[24]. In AKIN stage 2, there are 2 thrombocytopenic patients.Hence thrombocytopenia can be taken as a marker to predict severity of AKI. Also thrombocytopenia is directly proportional to severity of AKI .[25]

ASV VIALS REQUIRED TO NEUTRALISE IN RELATION TO AKIN STAGING:

In patient who required 10vials , only 17% developed AKI. Inj.ASV was not needed to be repeated in these patients, it is sign of mild-moderate envenomation.Patients requiring low dose ASV is associated.with better prognosis[26].In patients requiring 11-20 vials of ASV, 24 patients didn't develop AKI and 56 patients developed AKI, it is a sign of moderate to severe envenomation. In third group, who required. 21-30 vials of ASV, 38 patients among 41means 92% developed severe AKI. Some patients required blood products like FFP, Whole blood in addition to Inj.ASV. Maximum ASV vials given in these patient was 30 vials.

INTERVENTIONS NEEDED RELATED TO AKIN STAGING :

In our study among 150 patients, 99 patients developed acute kidney injury. Among them 65 patients are in AKIN 1 stage and 1 death occured and other patients recovered completely without in need of dialysis. 13 Patients developed AKIN stage 2, among them no one required dialysis and all of them recovered completely . 21 patients developed AKIN stage 3. Among them 19 patients required dialysis and 4 patients died .Most of the patient, 90% patient required dialysis and some of them died in spite of dialysis and effective management. Late presentation may be one of the reason for worst prognosis.

Acute tubule interstitial necrosis is the most common cause acute Kidney injury.

Diffuse cortical necrosis is having worst prognosis, patient will become dialysis dependant, will require Renal transplant[27]

OUTCOME OF PATIENT :

In our study, 99 patients developed AKI. 92% of patients recoveredcompletely. 64 patients with AKIN 1, 13 patients with AKIN2, 1 patients with AKIN 3 recovered with conservative management. 7 patients become Dialysis dependent for more than a month and progressed to chronic kidney Disease and advised to monitor renal parameters regularly and 5 patient died in hospital, 4 of them in stage3 AKIN and one person in stage 1 AKIN.

Early administration of Inj.ASV, adequate hydration, management of cellulitis are the important factors for good outcome

CONCLUSION

1) A severe acute renal damage is highly linked with a delay in bringing the patient to the hospital.
2) Poor prognosis is seen in patients with rapidly progressing cellulitis and those who have coagulopathy.
3) A poor prognosis is closely associated with moderate to serve thrombocytopenia.
4) Elderly patients with previous co morbidities like systemic hypertension and diabetes are predisposed in developing acute kidney injury and poor prognosis.
5) Urine output, serial renal parameters, should be monitored as they decides the prognosis.
6) AKIN staging can be utilised for early diagnosis, treatment, and forecast the prognosis and outcome.
7) Snake bite complications may be avoided with prompt treatment and acute kidney injury in young, healthy, working people, the morbidity and death can be prevented by effective management.

SUMMARY:

This study was conducted in Government Chengalpattu Medical College Hospital, with study population 150, with aim to predict the poor outcomes of acute kidney injury in patients with signs and symptoms of envenomation.

Information was gathered about the circumstances of the bite, the type of snake it was, first aid, initial therapy, the length of time it took to get to the hospital, co-morbidities, and vital signs. Basic Renal Parameters, WBCT , and complete hemogram are essential to know the outcomes and prognosis. Site of envenomation and systemic envenomation characteristics recorded.

Urine output, 8^{th} hourly renal parameters and daily CBC, were monitored and documented. Patients were classified into 4 Groups – NO AKI, AKIN 1, AKIN 2, AKIN 3. Management was done with adequate IV Fluids, Inj.ASV, Dialysis and treatment of local cellulitis.All have been documented with a proforma for this study.

Observations and results were analysed. Incidence of acute kidney injury in my study is 66 %. 21% are in stage 3 AKIN and most of them required dialysis. Few became Dialysis Dependant due to cortical necrosis, most of them are known case of Hypertension.

Case fatality rate in our study is 3.3 %. Among them four developed stage 3 and one developed stage 1 AKIN. Death was due to complications of Acute Kidney Injury.

Early hospital admission, treatment with Inj.ASV, appropriate hydration, monitoring of renal parameters, and simple bedside 20-minute WBCT monitoring may prevent unnecessary snake bite-related morbidity and mortality in healthy, young populations.

BIBLIOGRAPHY

1. Bawaskar HS. “Snake Bite Poisoning”, Chapter 59, API text book of Medicine 2007: 349
2. David A Warrell : WHO Guidelines for the management of snake-bites For South East Asian Countries. 2010
3. Warrel DA, Injuries, envenoming, poisoning and allergic reactions caused by animals. In: Wealtherall DJ, Ledingham JGG, Warrell DA (eds) Oxford Textbook of Medicine. 2nd edn. Oxford, Oxford University Press. 1987;6.66-6.77.
4. Warrel D.A. Injuries, enenoming, poisoning and allergic reactions caused by animals, Ed. Weatherall OJ., Ledingham J.G.G., Warrel .D.A: Text Book of Medicine 3rd Edition, Vol. I, Oxford University Press,1996.
5. Naing S. Clinical profile of viper bite cases. Divisional Hospital, Magwe (1981-82). Burmese Medical Journal1985;31:195-203.
6. BANERJEE R. N. Poisonous snakes in India, their venoms symptomatology and treatment of envenomation. In: Progress in clinical medicine in India. 1st ed, M .M. S. Abuja, Ed. (Amold-Heinman Publishers, New Delhi) 1978 p.136 - 177.
7. Reid HA. Animal Poisons. In: Manson Bahr PEC, Apted FIC, (Eds). Manson's Tropical Diseases, 18th edition. London. Balliere-Tindall 1982;544-546.
8. Saini RK, Sharma S, Singh S, Pathania NS. Snake bite poisoning: A preliminary report. J AssocPhys India 1984; 32(2):195-197

9. Hati A.K. et al: Epidemiology of snake bite in the district of Burdan (West Bengal), J. of Indian Medical Association - 1992 Jan. 90 (6): p. 145-7.
10. Manson - Bhar P.H: Animal poisons, 'Mansons' Tropical Diseases, 15th Edition, Cassel, London -1960.
11. Sharma N, Chauhan S, Faruqi S, Bhat P, Varma S; Snake envenomation in a north Indian hospital. Emerg Med J. 2005; 22:118-20
12. BROOK V. B. 1971. Quoted from snake venoms and envenomation. Ed Sherman A. Minton. Pub. Marcekk Decker Inc., New York,p.121.
13. REID H. A. 1963 In Venomous Animals and their venoms. EdsW.Bucheni, E.Buckley and V.Deulofeu. 611 - 641. (London and New York; Academic Press)vol-l.
14. ACHARYA V.N, METHAP.J,PATELM, CHAW ALA K.P et. AI. 1981 "Primary excessive fibrinolysis following snakebite. A case report". J-Assoc-Physician-India 29:577.
15. . Baglin T: Disseminated intravascular coagulation, diagnosis and management, British Medical Journal, March - 1996, 312 (683 -7).
16. aul VK. Animal and insect bites. In: Singh M (Ed). Medical Emergencies in Children. 2nd ed. New Delhi; Sagar Publications,1993
17. . CHERIAN A. M, TARIANG D. et At. Jan 1999 "A 145 Randomized Controlled Trail To Compare The Effectiveness Of A Lower Dose Versus Conventional Dose Of Snake AntiVenom, In Cases Of Snake Bite With Systemic Envenomation J-Assoc-Physician-India, Vol. 47, No 1:63.

18. Athappan G, Balaji MV, Navaneethan U. Acute renal failure in snake envenomation: a large prospective study. Saudi J Kidney Dis Transpl, 2008; 19(3):404-10.
19. Chetan, P.R., et al. "Incidence and clinical features of snakebite cellulitis at Kims, Hubli." Journal of Evolution of Medical and Dental Sciences, vol. 3, no. 71, 18 Dec. 2014, pp. 15070+. Gale Academic OneFile,
20. Kohli HS, Sakhuja V, Snake bites and acute renal failure Saudi J Kidney Dis Transpl 2003 14(2):165-76.
21. Jayanta Paul A: Haemorrhagicriecrotizing and edema forming effects of snake venom, Edited by Lee C V Berlin. Sevinger - Verlag - 1979, p. 480 - 546.
22. Suchithra N, Pappachan JM, Sujathan P; Snakebite envenoming in Kerala, South India: Clinical profile and factors involved in adverse outcomes. Emerg, 30 (50348-53) Med J. 2008; 25:200– 4.
23. Narvencar K; Corralation between timing of ASV administration and complications in snake bites. J Assoc Physicians India 2006; 54: 717-9
24. Isbister GK, Little M, Cull G, McCoubrie D, Lawton P, Szabo F, et al. Thrombotic microangiopathy from Australian brown snake (Pseudonaja) envenoming. Intern Med J 2007;37:523-8. Back to cited text no. 8
25. Chug K.S: Snakebite induced acute renal failure in India, KidneyInternational - 1989m 35: p. 891 - 907.

26. Pandey PCBajaj SSrivastava A (**2016**) A Clinico-Epidemiological profile of neuroparalytic snake bite: using low dose ASV in a tertiary care centre from North India The Journal of the Association of Physicians of India 64:16–20.
27. Sarkhel SGhosh RMana KGantait K (**2017**) A hospital based epidemiological study of snakebite in Paschim Medinipur district, West Bengal, India Toxiology Reports 4:415–419.

STUDY PROFORMA:

" A STUDY ON PREDICTORS OF POOR OUTCOME IN SNAKE BITE INDUCED ACUTE KIDNEY INJURY AT CHENGALPATTU MEDICAL COLLEGE HOSPITAL"

Name:
IP. No:
Reg.No :
Age/sex:
Address:

D.O.A:
D.O.D:

DATE & TIME OF BITE
DATE & TIME OF ADMISSION
DELAY IN ADMISSION (HOURS)
TOURNIQUET APPL IED : YES / NO
SNAKE IDENTI F IED : YES / NO
I F YES
VIPER / COBRA / KRAIT / OTHERS

COMPLAINTS OF THE PATIENT:

1.Pain at the bite site : Yes / No
2. Bleeding from bite mark : Yes / No
3. Vomiting : Yes / No

PAST HISTORY :H/ O DM / HTN / SEIZURE DISORDER / COPD/ CAD / OTHERS

PERSONAL HISTORY :

DIET : Vegetarian / Non- Vegetarian / Mixed Diet

Smoking :

Alcoholism :

TREATMENT HISTORY :

Duration of treatment :

No. Of vials Inj. ASV administered :

Clotting time on Referral :

CLINICAL FEATURES

VITAL SIGNS AT THE TIME OF ADMISSION

BP

PULSE RATE

RESPIRATORY RATE

TEMPERATURE

SIGNS OF TOXICITY

SIGNS	**DAY 1**	**DAY 2**	**DAY 3**	**DAY 4**	**DAY 5**	**TILL DISCHARGE**
VITAL SIGNS **BP** **PULSE RATE**						
CELLULITIS						

REGIONAL LYMPHADENOPATHY						
CLOTTING TIME						
PTOSIS						
RESPIRATORY PARALYSIS						
SIGNS OF DIC						

SYSTEMIC EXAMINATION:

1. Cardiovascular System :

2.Respiratory system :

3. Per Abdomen :

4. Central Nervous System :

INVESTIGATIONS :

USG KUB :

OTHER INVESTIGATIONS (IF NEEDED) :

PARAMETERS	**DAY 1**	**DAY 2**	**DAY 3**	**DAY 4**	**DAY 5**	**TILL DISCHARGE**
B. UREA						
SERUM CREATININE						
INPUT OUTPUT (HRLY)						
HB PLATELET COUNT PCV						
NO. OF VIALS INJ.ASV ADMINISTERED						

ALLERGIC REACTIONS TO INJ.ASV						
TREATMENT: 1.CONSERVATIVE						
2.PERITONEAL DIALYSIS						
3.HEMODIALYSIS						

FINAL INFERENCE: SNAKE BITE WITH ..... TOXICITY WITH/

WITHOUT ACUTE KIDNEY INJURY (AKIN STAGE.....) MANAGED WTH.....

INFORMED CONSENT FORM

Title of the Study : " **A STUDY ON PREDICTORS OF POOR OUTCOME IN SNAKE BITE INDUCED ACUTE KIDNEY INJURY AT CHENGALPATTU MEDICAL COLLEGE HOSPITAL"**

Name of the Participant : _____.

Name of the Principal (Co-

Investigator):_____.

Name of the Institution: Chengalpattu Medical college and Hospital.

Name and address of the sponsor / agency (ies) (If any) :

_____

_____

____.

Documentation of the informed consent

I _____have read the information in this form (or it has been read to me). I was free to ask any questions and they have been answered. I am over 18 years of age and, exercising my free power of choice, hereby give my consent to be included as a participant in " **A STUDY ON PREDICTORS OF POOR OUTCOME IN SNAKE BITE INDUCED ACUTE KIDNEY INJURY AT CHENGALPATTU MEDICAL COLLEGE HOSPITAL"**

title of the study).

I have read and understood this consent form and the information provided to me.

I have had the consent document explained to me.

I have been explained about the nature of the study.

I have been explained about my rights and responsibilities by the investigator.

I have been informed the investigator of all the treatments I am taking or have taken in the past _____ months including any native (alternative) treatment.

I have been advised about the risks associated with my participation in this study.*

I agree to cooperate with the investigator and I will inform him/her immediately if I suffer unusual symptoms.*

I have not participated in any research study within the past _____month(s).*

I have not donated blood within the past _____months----add if the study involves extensive blood sampling.*

I am aware of the fact that I can opt out of the study at any time without having to give any reason and this will not affect my future treatment in this hospital.*

I am also aware that the investigator may terminate my participation in the study at any time, for any reason, without my consent.

I hereby give permission to the investigators to release the information obtained from me as result of participation in this study to the sponsors, regulatory authorities, Govt. agencies, and IEC. I understand that they are publicly presented.

I have understand that my identity will be kept confidential if my data are publicly presented .

I have had my questions answered to my satisfaction.

I have decided to be in the research study.

I am aware that if I have any question during this study, I should contact the investigator. By signing this consent form I attest that the information given in this document has been clearly explained to me and understood by me, I will be given a copy of this consent document.

signature of the investigator :

signature of the participant :

சுயஒப்புதல் படிவம்

ஆய்வுசெய்யப்படும் தலைப்பு : "செங்கல்பட்டு மருத்துவக் கல்லூரியில் பாம்பு கடியால் ஏற்படும் சிறுநீரக பிரச்சனைகளை முன்னரிவதற்கான சாத்தியக்கூறுகள்
பற்றிய ஆய்வு"

"

பங்குபெறுபவரின் வயது:

பங்குபெறுபவரின் எண் :

மேலேகுறிப்பிட்டுள்ள மருத்துவஆய்வின் விவரங்கள் எனக்கு விளக்கபட்டுள்ளது. நான் இவ்வாய்வில் தன்னிச்சையாக பங்கேற்கின்றேன். எந்தகாரணத்தினாலோ, எந்த சட்டசிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக்கொள்ளலாம் என்றும் அறிந்துகொண்டேன்.

இந்த ஆய்வு சம்பந்தமாகவோ, இதைசார்ந்து மேலும் ஆய்வுமேற்கொள்ளும்போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர், என்னுடைய மருத்துவஅறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவை இல்லை என அறிந்துகொள்கிறேன். இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ,முடிவையோ பயன்படுத்திக் கொள்ள மறுக்கமாட்டேன்.

இந்த ஆய்வில் பங்குகொள்ள ஒப்புக்கொள்கிறேன். இந்த ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம்:

சாட்சியாளரின் கையொப்பம்:

இடம்:

தேதி:

பங்கேற்பவரின் பெயர் மற்றும் விலாசம்:

ஆய்வாளரின் கையொப்பம் :

இடம்: தேதி:

KEY TO MASTER CHART

A/S	Age/sex
SOB	Site of bite
BNT	Bite to needle time
COM	Comorbidities
HTN	Hypotension
RF	Respiratory failure
CEL	Cellulitis
RL	Regional lymphadenopathy
WBCT	Whole blood clotting time
IC SR	Initial creatinine Serial creatinine
PLC	Platelet count
AKIN	Acute kidney injury network
CM	Conservative management
CR	Complete recovery
DD	Dialysis dependent

MASTER CHART

S.NO	NAME	AGE/SEX	SOB	BNT	COM	HTN	CELL	RL	WBCT	RF	ICR	S.VALUE	OUTPUT	PLC	AKIN	ASV	MX	OUTCOME
1	SUNDAR	37/M	LTFOOT	2 HRS	SHTN	NO	NO	YES	>20	NO	1.7	2.3	NON OLIGURIA	160000	1	28	CM	CR
2	JEYAPRAKASH	40/M	RT FOOT	15MIN	SHTN	NO	YES	YES	>20	NO	2	3.1	NON OLIGURIA	100000	1	24	CM	CR
3	RAMAYI	55/F	LTFOOT	3HRS	NO	NO	YES	NO	<20	NO	2.2	2.9	NON OLIGURIA	140000	1	10	CM	CR
4	RAJAAMMAL	60/F	LTFOOT	1 HR	DM	NO	YES	YES	>20	NO	3	3.3	OLIGURIA	80000	3	28	DIALYSIS	DD
5	GANDHIYAMMAL	80/F	RT FOOT	30MIN	NO	NO	YES	NO	>20	NO	2	2.5	NON OLIGURIA	190000	1	10	CM	CR
6	RAMESH	32/M	RT HAND	2HRS	NO	NO	NO	YES	>20	NO	2	4.9	OLIGURIA	250000	3	28	DIALYSIS	CR
7	VICKY	34/M	RT FOOT	6 HRS	NO	NO	YES	YES	>20	NO	1	4.5	OLIGURIA	75000	3	30	DIALYSIS	CR
8	CHINNAPILLAI	50/M	RT ANKLE	8 HRS	NO	NO	NO	NO	>20	NO	1	1.4	NON OLIGURIA	160000	1	16	CM	CR
9	SAROJA	65/F	LTFOOT	6HRS	NO	NO	YES	YES	>20	NO	2	2.7	NON OLIGURIA	280000	1	24	CM	CR
10	KUPPAN	45/M	RT HAND	5HRS	HTN	NO	YES	NO	<20	NO	1.5	2	NON OLIGURIA	160000	1	10	CM	CR
11	MURUGAN	45/M	RT FOOT	2 HRS	NO	NO	NO	NO	>20	NO	2	2.5	NON OLIGURIA	170000	1	28	CM	CR
12	LAKSHMI	65/F	RT HAND	1 HR	NO	NO	YES	YES	>20	NO	1	1.5	NON OLIGURIA	250000	1	24	CM	CR
13	KUPPUSWAMY	70/M	LTANKLE	2 HRS	NO	NO	NO	NO	>20	NO	1	2.1	NON OLIGURIA	370000	1	16	CM	CR
14	AMUTHA	20/F	LTANKLE	5HRS	NO	NO	YES	NO	>20	NO	1	1.5	NON OLIGURIA	450000	1	20	CM	CR
15	RAMAN	25/M	RT FOOT	2HRS	NO	NO	NO	NO	<20	NO	3	3.2	NON OLIGURIA	350000	1	16	CM	CR
16	PERIYASAMY	65/M	LTFOOT	4 HRS	NO	NO	YES	YES	>20	NO	1	3.9	NON OLIGURIA	80000	3	24	CM	CR
17	SIDDAN	70/M	RT FOOT	3HRS	NO	NO	NO	NO	>20	NO	1	1.5	NON OLIGURIA	450000	1	16	CM	CR
18	PATCHAIYAMAL	40/F	LTANKLE	2HRS	NO	NO	NO	NO	>20	NO	1	2	NON OLIGURIA	190000	2	16	CM	CR
19	RAVI	40/M	RT ANKLE	3HRS	NO	NO	YES	YES	>20	YES	1	2.5	NON OLIGURIA	459000	2	28	CM	CR
20	NAMAGIRI	30/M	LTFOOT	4HRS	NO	NO	YES	NO	>20	NO	2	2.9	NON OLIGURIA	150000	1	28	CM	CR
21	SAROJA	54/F	LTANKLE	5HRS	SHTN	NO	NO	NO	>20	NO	1	2.5	NON OLIGURIA	580000	2	30	CM	CR
22	BACKIYAM	45/F	RT FOOT	4HRS	NO	NO	YES	YES	>20	NO	2	2.2	NON OLIGURIA	160000	0	16	CM	CR
23	PRAKASH	18/M	LTFOOT	5HRS	PLHA	NO	YES	NO	<20	NO	2	2.5	NON OLIGURIA	540000	1	18	CM	CR
24	SANKAR	34/M	RT HAND	2 HRS	SHTN	NO	NO	NO	>20	NO	1	2.9	NON OLIGURIA	190000	2	30	CM	CR
25	SENTHIL	50/M	LTANKLE	3HRS	NO	NO	NO	NO	>20	NO	1	1.3	NON OLIGURIA	250000	0	20	CM	CR
26	DIVYA	39/F	RT FOOT	4HRS	NO	NO	YES	YES	<20	NO	1	1.6	NON OLIGURIA	85000	1	20	CM	CR
27	SARAVANAN	21/M	LTFOOT	4HRS	NO	NO	NO	NO	>20	NO	1	1.5	NON OLIGURIA	480000	0	20	CM	CR
28	RAMAN	30/M	RT FOOT	3 HRS	NO	NO	NO	NO	>20	NO	1	2	NON OLIGURIA	548200	2	30	CM	CR

29	KANMANI	45/F	LTANKLE	3HRS	SHTN/DM	NO	YES	YES	<20	NO	3.2	4.5	NON OLIGURIA	470000	1	28	CM	CR
30	MANI	50/M	RT ANKLE	2HRS	NO	NO	NO	NO	>20	NO	1	1.5	NON OLIGURIA	150000	1	16	CM	CR
31	RAMANI	52/F	LTFOOT	2.5HRS	SHTN/DM	YES	YES	NO	>20	YES	1	1.8	NON OLIGURIA	250000	1	30	MV	DEATH
32	SARASWATI	32/F	RT FOOT	1HR	NO	NO	YES	YES	>20	YES	1.2	1.9	NON OLIGURIA	150000	1	30	CM	CR
33	KANNAN	52/M	RT HAND	4 HRS	NO	NO	YES	YES	>20	NO	2	2.5	NON OLIGURIA	450000	1	20	CM	CR
34	SAGAR	61/M	RT FOOT	3HRS	NO	NO	YES	YES	>20	NO	1	1.3	NON OLIGURIA	250000	0	30	CM	CR
35	PACHAI	37/M	LFHAND	6HRS	NO	NO	YES	NO	>20	NO	1	1.6	NON OLIGURIA	340000	1	20	CM	CR
36	NATHIYA	25/F	LTANKLE	5HRS	NO	NO	NO	NO	>20	NO	1.2	1.5	NON OLIGURIA	240000	0	20	CM	CR
37	MARAPAN	45/M	RT FOOT	2HRS	NO	NO	NO	NO	>20	NO	1	1.2	NON OLIGURIA	540000	0	20	CM	CR
38	MARI	34/M	LTHAND	4HRS	NO	NO	YES	YES	<20	NO	1	1.5	NON OLIGURIA	450000	1	20	CM	CR
39	SUJATHA	45/F	RT FOOT	1 HR	NO	NO	NO	NO	>20	NO	1	1.3	NON OLIGURIA	98000	0	24	CM	CR
40	RAJAMANI	60/M	RT ANKLE	5HRS	SHTN	NO	YES	YES	>20	NO	2	6.9	NON OLIGURIA	150000	3	30	DIALYSIS	DD
41	RAMASWAMY	42/M	RT FOOT	2 HRS	NO	NO	YES	YES	>20	NO	1.2	3	NON OLIGURIA	450000	2	16	CM	CR
42	VELAN	50/M	LTHAND	3HRS	NO	NO	YES	YES	>20	NO	1	1.2	NON OLIGURIA	250000	0	20	CM	CR
43	PALANI	26/M	RT FOOT	1HR	NO	NO	YES	YES	>20	NO	0.9	1.4	NON OLIGURIA	300000	1	16	CM	CR
44	THANAM	29/F	RT ANKLE	5 HRS	NO	NO	YES	YES	<20	NO	1	1.5	NON OLIGURIA	260000	1	20	CM	CR
45	KANDHAN	45/M	LTHAND	1 HR	NO	NO	YES	YES	>20	NO	2	6.2	OLIGURIA	290000	3	28	DIALYSIS	CR
46	GOVIND	34/M	RT FOOT	2HRS	NO	NO	YES	YES	>20	NO	1	1.3	NON OLIGURIA	260000	1	16	CM	CR
47	VINAY	35/M	LFHAND	6HRS	NO	NO	YES	NO	<20	NO	1	1.2	NON OLIGURIA	540000	0	16	CM	CR
48	VIJAY	34/M	RT HAND	2HRS	NO	NO	YES	NO	<20	NO	0.9	1.2	NON OLIGURIA	156000	0	20	CM	CR
49	PAPAN	52/F	LTFOOT	1.5 HRS	NO	NO	YES	NO	>20	NO	1.2	1.3	NON OLIGURIA	160000	0	16	CM	CR
50	VALLI	45/F	RT FOOT	5HRS	NO	NO	YES	YES	>20	NO	1	1.5	NON OLIGURIA	180000	1	20	CM	CR
51	SARASWATI	42/F	LTHAND	2 HRS	NO	NO	YES	NO	<20	NO	1	1.3	NON OLIGURIA	450000	1	16	CM	CR
52	NALLAN	34/M	RT ANKLE	5HRS	NO	NO	YES	YES	>20	NO	0.8	1	NON OLIGURIA	156000	0	20	CM	CR
53	KANNI	45/F	RT FOOT	4HRS	NO	NO	YES	YES	>20	NO	1.2	1.6	NON OLIGURIA	160000	1	16	CM	CR
54	SELVI	56/F	LTANKLE	6HRS	DM	NO	YES	YES	>20	NO	1.4	4.5	OLIGURIA	80000	3	24	DIALYSIS	CR
55	CHINNAYA	43/M	RT FOOT	30 MIN	NO	NO	YES	YES	<20	NO	0.8	1	NON OLIGURIA	450000	0	20	CM	CR
56	AYAMAL	57/F	LTFOOT	2 HR	NO	NO	YES	YES	>20	NO	1.2	1.3	NON OLIGURIA	460000	0	16	CM	CR
57	RASATHI	23/F	RT HAND	2HRS	NO	NO	YES	YES	<20	NO	1	1.1	NON OLIGURIA	250000	0	20	CM	CR

58	SOUNDARYA	43/F	RT LEG	1.5HRS	NO	NO	YES	YES	>20	NO	1.2	1.4	NON OLIGURIA	420000	0	16	CM	CR
59	NANDHAN	32/M	LTLEG	3HRS	NO	NO	YES	YES	>20	NO	1	2.4	NON OLIGURIA	180000	2	28	CM	CR
60	GOVINDHAN	44/M	LTHAND	5HRS	NO	NO	YES	NO	<20	NO	1.1	1.5	NON OLIGURIA	240000	1	16	CM	CR
61	VADIVU	55/F	LTFOOT	7HRS	SHTN/DM	YES	YES	YES	>20	NO	1.3	5.4	OLIGURIA	45000	3	30	DIALYSIS	CR
62	ELAVARASI	45/F	RT HAND	2 HRS	NO	NO	YES	YES	>20	NO	1	1.2	NON OLIGURIA	250000	0	20	CM	CR
63	VALLIAMAL	65/F	LTANKLE	5HRS	SHTN	YES	YES	YES	>20	YES	1.5	5.2	OLIGURIA	80000	3	30	DIALYSIS	DEATH
64	KUMAR	55/M	RT FOOT	2.5HRS	NO	NO	NO	NO	>20	NO	0.8	1.2	NON OLIGURIA	150000	0	10	CM	CR
65	MEERA	62/F	RT ANKLE	6HRS	DM	NO	YES	YES	>20	YES	1.9	5.6	OLIGURIA	45000	3	30	DIALYSIS	DEATH
66	MUTHU	22/M	LEFTARM	4HRS	NO	NO	NO	NO	>20	NO	0.8	1.1	NON OLIGURIA	190000	0	10	CM	CR
67	LAVANYA	22/F	LEFT SHOULDER	1.5HRS	NO	NO	NO	NO	>20	NO	0.9	1.2	NON OLIGURIA	180000	0	10	CM	CR
68	ARUN	54/M	RT FOOT	3 HRS	NO	NO	YES	YES	>20	NO	1.5	2	NON OLIGURIA	540000	1	16	CM	CR
69	GOVIND	45/M	LTARM	30 MIN	SHTN	NO	NO	NO	>20	NO	0.8	1.3	NON OLIGURIA	184000	0	10	CM	CR
70	SALEEM	23/M	RT FOOT	1 HR	NO	NO	NO	YES	>20	NO	1	1.5	NON OLIGURIA	450000	0	10	CM	CR
71	RAMAN	44/M	LTHAND	2 HRS	DM	NO	YES	YES	>20	NO	1.3	1.9	NON OLIGURIA	156000	1	16	CM	CR
72	SARASWATHI	56/F	LTFOOT	6HRS	SHTN	NO	YES	YES	>20	NO	2.3	6.2	OLIGURIA	54000	3	30	DIALYSIS	DD
73	SEETHA	34/F	RT FOOT	2 HRS	NO	NO	NO	NO	<20	NO	1.2	1.4	NON OLIGURIA	265000	0	16	CM	CR
74	VENKAT	45/M	LTFOOT	1.5HRS	NO	NO	NO	NO	<20	NO	0.9	1.4	NON OLIGURIA	165000	0	10	CM	CR
75	RAMASWAMY	70/M	RT HAND	2HRS	DM/SHTN	NO	YES	YES	>20	NO	1.5	2.6	NON OLIGURIA	120000	1	16	CM	CR
76	PERUMAL	24/M	LTHAND	3 HRS	NO	NO	NO	NO	<20	NO	0.9	1.3	NON OLIGURIA	154000	0	10	CM	CR
77	SELVAM	34/M	RT HAND	30 MIN	SHTN	NO	NO	NO	<20	NO	1.2	1.3	NON OLIGURIA	560000	0	10	CM	CR
78	PADMA	45/F	LTFOOT	1 HR	NO	NO	YES	YES	>20	NO	1.5	2.3	NON OLIGURIA	165000	1	16	CM	CR
79	ROSHAN	18/M	RT HAND	45 MIN	NO	NO	YES	YES	>20	NO	0.9	1.2	NON OLIGURIA	150000	0	16	CM	CR
80	RAMESH	52/M	LTFOOT	4 HRS	DM/SHTN	YES	YES	YES	>20	YES	1.9	6.2	OLIGURIA	54000	3	30	MV	DEATH
81	JAYA	36/F	RT HAND	2 HRS	NO	NO	NO	NO	<20	NO	0.8	1.4	NON OLIGURIA	189000	0	10	CM	CR
82	PRASAD	72/M	LEFTARM	1.5 HRS	SHTN	NO	YES	YES	>20	NO	1.5	2.6	NON OLIGURIA	90000	1	16	CM	CR
83	KANNAN	54/M	RT HAND	45 MIN	NO	NO	NO	NO	<20	NO	0.9	1.4	NON OLIGURIA	198000	0	10	CM	CR
84	KAVYA	25/F	LTHAND	1 HR	NO	NO	YES	NO	<20	NO	0.8	1.3	NON OLIGURIA	80000	0	20	CM	CR
85	DHINA	34/M	RT FOOT	1.5 HRS	NO	NO	YES	YES	>20	NO	1.5	2.4	NON OLIGURIA	160000	1	16	CM	CR
86	PALANIAPPAN	29/M	RT HAND	2 HRS	SHTN	NO	YES	YES	>20	NO	1	1.2	NON OLIGURIA	190000	0	20	CM	CR

87	VENNILA	45/F	LTANKLE	30MIN	NO	NO	YES	YES	>20	NO	1.5	3.2	NON OLIGURIA	90000	2	28	CM	CR
88	SELVI	34/F	RT FOOT	1HR	NO	NO	NO	NO	<20	NO	1	1.1	NON OLIGURIA	450000	0	10	CM	CR
89	RAMESH	45/M	LTHAND	2 HRS	DM	NO	YES	YES	>20	NO	1.3	2.2	NON OLIGURIA	160000	1	16	CM	CR
90	DEVI	50/F	LTTHIGH	1 HR	DM/SHTN	NO	YES	YES	<20	NO	0.9	1	NON OLIGURIA	120000	0	16	CM	CR
91	VIGNESH	18/M	RT FOOT	1.5 HRS	NO	NO	NO	YES	<20	NO	0.8	1.1	NON OLIGURIA	160000	0	10	CM	CR
92	YAMUNA	69/F	LTANKLE	5HRS	SHTN	NO	YES	YES	>20	NO	2.4	6.2	OLIGURIA	75000	3	30	DIALYSIS	DD
93	RAMANI	62/F	RT THIGH	4.5HRS	DM/SHTN	NO	YES	YES	>20	YES	1.9	5.2	OLIGURIA	120000	3	28	DIALYSIS	CR
94	BABU	45/M	RT HAND	2HRS	NO	NO	NO	YES	<20	NO	1.1	0.9	NON OLIGURIA	540000	0	10	CM	CR
95	SELVAM	28/M	RT FOOT	1.5HRS	NO	NO	NO	NO	>20	NO	0.9	1.2	NON OLIGURIA	160000	0	10	CM	CR
96	RASHIKA	36/F	LTHAND	30 MIN	SHTN	NO	NO	NO	<20	NO	1.1	1	NON OLIGURIA	540000	0	10	CM	CR
97	YUVARAJ	49/M	LTFOOT	6HRS	DM	NO	YES	YES	>20	NO	1.6	5.9	OLIGURIA	89000	3	30	DIALYSIS	DD
98	RAVI	68/M	RT FOOT	4HRS	SHTN	NO	YES	YES	>20	YES	2.6	6.9	OLIGURIA	156000	3	30	DIALYSIS	CR
99	LAVANYA	28/F	RT HAND	2HRS	NO	NO	NO	NO	<20	NO	0.8	1	NON OLIGURIA	450000	0	10	CM	CR
100	BOOBALAN	45/M	LTHAND	1.5HRS	DM	NO	NO	YES	<20	NO	1.1	1.3	NON OLIGURIA	160000	0	10	CM	CR
101	SASIKALA	50/F	RT FOOT	7HRS	NO	NO	YES	YES	>20	NO	2.6	6.4	OLIGURIA	120000	3	30	DIALYSIS	CR
102	DILLIBABU	46/M	LTTHIGH	5HRS	DM	YES	YES	YES	>20	YES	1.4	1.5	NON OLIGURIA	190000	0	28	MV	CR
103	RAJA	23/M	RT HAND	2HRS	NO	NO	NO	NO	<20	NO	0.9	1	NON OLIGURIA	180000	0	10	CM	CR
104	RAVINDRAN	40/M	RT FOOT	3 HRS	NO	NO	YES	NO	<20	NO	0.9	1	NON OLIGURIA	160000	0	20	CM	CR
105	RAMASWAMY	32/M	RT HAND	2HRS	NO	NO	NO	NO	>20	NO	0.8	1.2	NON OLIGURIA	169000	0	16	CM	CR
106	DIVYA	18/F	RT FOOT	1 HR	NO	NO	NO	NO	<20	NO	0.7	1	NON OLIGURIA	450000	0	10	CM	CR
107	GOVIND	69/M	RT FOOT	8HRS	DM	NO	YES	YES	>20	YES	2.9	6.8	OLIGURIA	56000	3	30	DIALYSIS	DEATH
108	RAMADOSS	57/M	RT HAND	1 HR	SHTN/DM	NO	YES	YES	>20	NO	1.5	1.6	NON OLIGURIA	450000	1	16	CM	CR
109	VIGNESH	28/M	LTFOOT	2 HRS	NO	NO	NO	NO	<20	NO	0.8	1	NON OLIGURIA	150000	0	10	CM	CR
110	DIVYA	52/F	LTHAND	1 HR	DM	NO	NO	NO	>20	NO	1.9	3	NON OLIGURIA	480000	1	20	CM	CR
111	RAMYA	45/F	RT FOOT	2 HRS	SHTN	NO	NO	YES	>20	NO	0.9	1.1	NON OLIGURIA	120000	0	20	CM	CR
112	BANUPRIYA	34/F	RT HAND	1 HR	NO	NO	YES	NO	>20	NO	0.9	1.2	NON OLIGURIA	150000	0	16	CM	CR
113	KARAN	55/M	RT FOOT	10 HRS	DM	NO	YES	YES	>20	NO	1.9	6.4	NON OLIGURIA	98000	3	30	DIALYSIS	DD
114	ANBUMANI	34/M	RT ARM	2 HRS	NO	NO	NO	NO	<20	NO	0.9	1.1	NON OLIGURIA	189000	0	10	CM	CR
115	RAVICHANDRAN	54/M	LTFOOT	3 HRS	DM	NO	NO	NO	>20	NO	0.9	1.6	NON OLIGURIA	192000	1	10	CM	CR

116	LAKSHMI	65/F	RT ARM	2 HRS	SHTN	NO	YES	YES	>20	NO	1.4	2.9	NON OLIGURIA	160000	2	28	CM	CR
117	RAMANI	45/F	LTFOOT	1 HR	DM	NO	NO	YES	<20	NO	0.8	1.4	NON OLIGURIA	189000	1	16	CM	CR
118	DEVA	34/M	RT HAND	3.5HRS	NO	NO	YES	YES	>20	NO	1.5	3	NON OLIGURIA	98000	2	16	CM	CR
119	MASILAMANI	29/M	LTFOOT	30 MIN	NO	NO	YES	YES	<20	NO	0.9	1	NON OLIGURIA	540000	0	10	CM	CR
120	NALLAN	45/M	RT HAND	45 MIN	DM	NO	YES	NO	<20	NO	1.2	1.4	NON OLIGURIA	489000	0	10	CM	CR
121	MADHU	35/F	RT FOOT	1 HR	NO	NO	NO	NO	<20	NO	0.9	1.2	NON OLIGURIA	450000	0	10	CM	CR
122	RAMANAN	31/M	LTFOOT	1.5 HRS	NO	NO	YES	YES	>20	NO	1.1	1.4	NON OLIGURIA	156000	1	20	CM	CR
123	KRISHNA	56/M	RT HAND	5 HRS	DM/SHTN	NO	YES	YES	>20	NO	2.6	6.9	NON OLIGURIA	60000	3	30	DIALYSIS	DD
124	VEERASAMY	45/M	LTFOOT	2 HRS	NO	NO	YES	YES	>20	NO	1.5	1.9	NON OLIGURIA	120000	1	20	CM	CR
125	RADHAN	32/M	LTHAND	30 MIN	NO	NO	NO	YES	>20	NO	1.1	1.4	NON OLIGURIA	169000	1	16	CM	CR
126	RAVI	56/M	RT FOOT	1 HR	DM	NO	YES	YES	>20	NO	1	1.9	NON OLIGURIA	189000	2	20	CM	CR
127	SELVI	54/F	LTHAND	1.5 HRS	NO	NO	YES	YES	>20	NO	1.4	2.6	NON OLIGURIA	450000	1	28	CM	CR
128	TAMIL	28/M	LTFOOT	3 HRS	NO	NO	YES	YES	>20	NO	1.2	1.6	NON OLIGURIA	136000	1	20	CM	CR
129	LAVANYA	45/F	RT HAND	4 HRS	NO	NO	YES	YES	<20	NO	1.1	1.5	NON OLIGURIA	250000	1	10	CM	CR
130	PRADEEP	34/M	LTHAND	3HRS	NO	NO	YES	YES	>20	NO	1.5	2.4	NON OLIGURIA	360000	1	16	CM	CR
131	RAMU	29/M	Rt hand	2 HRS	NO	NO	YES	YES	>20	NO	1.2	1.6	NON OLIGURIA	134000	1	16	CM	CR
132	PRADEEP	30/M	RT FOOT	3HRS	NO	NO	YES	YES	>20	NO	1.1	1.5	NON OLIGURIA	156000	1	20	CM	CR
133	JANSY	42/F	LEFTLEG	1 HR	NO	NO	NO	YES	>20	NO	1.4	1.9	NON OLIGURIA	134000	1	16	CM	CR
134	ETTIRAM	54/M	LTHAND	2HR	DM	NO	YES	YES	>20	NO	1.5	2.2	NON OLIGURIA	560000	1	20	CM	CR
135	JAMUNA	56/F	LTARM	4HR	NO	NO	YES	YES	>20	NO	2.2	5.6	OLIGURIA	98000	3	30	DIALYSIS	CR
136	SINDHU	29/F	RT FOOT	4.5HR	DM/SHTN	NO	YES	YES	>20	NO	1.4	1.7	NON OLIGURIA	120000	1	16	CM	CR
137	PALLAVI	45/F	RT ARM	4 HR	NO	NO	YES	YES	<20	NO	1.3	1.9	NON OLIGURIA	450000	1	20	CM	CR
138	LEELA	45/F	LTFOOT	2 HR	NO	NO	YES	YES	<20	NO	1.5	1.9	NON OLIGURIA	190000	1	16	CM	CR
139	SAM	34/M	RT ARM	3HR	SHTN	NO	YES	YES	<20	NO	1.4	2.1	NON OLIGURIA	320000	1	20	CM	CR
140	YOGIBABU	38/M	RT HAND	1.5 HR	NO	NO	YES	YES	<20	NO	1.2	1.7	NON OLIGURIA	210000	1	16	CM	CR
141	SARAVANAN	28/M	LTFOOT	3 HR	DM	NO	YES	YES	<20	NO	1.4	1.8	NON OLIGURIA	230000	1	16	CM	CR
142	JEEVA	56/M	LTARM	3.5HR	NO	NO	YES	YES	>20	NO	1.2	1.9	NON OLIGURIA	349000	1	16	CM	CR
143	JOHNSON	48/M	RT ARM	4HR	NO	NO	YES	YES	<20	NO	1	1.7	NON OLIGURIA	240000	1	16	CM	CR
144	LAKSHMI	35/F	LTFOOT	1.5 HR	DM	NO	YES	YES	>20	NO	1.2	2.4	NON OLIGURIA	230000	2	16	CM	CR

145	SANDHYA	45/F	RT ARM	2 HR	DM	NO	YES	YES	<20	NO	1.5	1.9	NON OLIGURIA	340000	1	16	CM	CR
146	AKITHA	48/F	RT LEG	1HR	SHTN	NO	YES	YES	<20	NO	1.2	2.2	NON OLIGURIA	450000	1	20	CM	CR
147	LALITHA	36/F	LTHAND	4 HR	NO	NO	YES	YES	<20	NO	1.3	2.1	NON OLIGURIA	340000	1	16	CM	CR
148	ASWIN	36/M	RT ARM	3 HR	SHTN	NO	YES	YES	<20	NO	1.4	2.4	NON OLIGURIA	230000	2	16	CM	CR
149	NARESH	56/M	LTHAND	5HR	NO	NO	YES	YES	<20	NO	1.7	2.3	NON OLIGURIA	340000	1	20	CM	CR
150	LOKESH	52/M	LTFOOT	3HR	NO	NO	YES	YES	<20	NO	1.5	1.9	NON OLIGURIA	450000	1	16	CM	CR