CARDIAC INVOLVEMENT IN SNAKE ENVENOMATION

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CERTIFICATE

This is to certify that the dissertation titled **"CARDIAC INVOLVEMENT IN SNAKE ENVENOMATION"** is the bonafide originalwork of **Dr.VINOTH KUMAR.S.,** in partial fulfillment of the requirements for M.D. Branch–I(General Medicine) Examination of the Tamilnadu Dr. M.G.R Medical University to be held in APRIL 2012. The Period of study was from May 2011 to November 2011.

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DECLARATION

I hereby solemnly declare that the dissertation titled **"CARDIAC INVOLVEMENT IN SNAKE ENVENOMATION"** was done by me at Madras Medical College and Rajiv Gandhi Government General Hospital, Chennai-3 during May 2011 to November 2011 under the guidance and supervision of my unit Chief Prof. C.Rajendiran, M.D.

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INTRODUCTION

Snake bite is an occupational disease of farmers, plantation workers, herdsmen, fishermen, snake workers and other food producers. It is therefore a medical problem that has important implications for the nutrition and economy of the countries where it occurs commonly.

Despite its importance, there have been fewer proper clinical studies of snake bite than of almost any other tropical disease. Snakebites probably cause more deaths in the region than do Entamoeba histolytica infections but only a small fraction of the research investment in amoebiasis has been devoted to the study of snake-bite.It would be better if governments, academic institutions, pharmaceutical, agricultural and other industries and other funding bodies,actively encourage and sponsor properly designed clinical studies of all aspects of snake-bite.

Snake Bites is a major health problem in India. India alone contributes to about 81,000 envenomations and 11,000 deaths annually. In Tamilnadu envenomations occur commonly due to snakes of Elapidae and Viperidae families causing local and systemic manifestations.

Cardiotoxicity has been recognized as a feature of snake envenoming. However, it is the neuromuscular paralysis and the respiratory failure with elapid bites and the coagulation abnormalities in viperine bites, which dominate treatment efforts in patients.

Cardiac involvement has not been fully studied earlier and so the present study is undertaken. This study is likely to contribute to the importance of assessing the cardiac status in snake envenomation.

AIMS AND OBJECTIVES

SIGNIFICANCE OF RESEARCH

To bring out the cardiac involvement in snake envenomation and to provide additional insights for management.

RESEARCH QUESTIONS

1) How many persons with snake bite have cardiac involvement?

2) What are the patterns of cardiac involvement?

3) Whether cardiac involvement has any clinical significance?

4) Do the patients with cardiac involvement need any special care.

OBJECTIVES

1) To find out the pattern of cardiac involvement in Snake Envenomation.

2) To elicit the Electrocardiographic abnormalities and functional status of heart during snake envenomation.

3) To analyse the variations in cardiac involvement with reference to different species of snakes.

4) To identify the importance of biomarkers in recognizing cardiac involvement.

REVIEW OF LITERATURE[1]

There are more than 3000 species of snakes in the world. For the purpose of clinical practice, snakes are classified into poisonous (venomous) and non-poisonous (non venomous) snakes. Poisonous snakes are classified into three families and they are

- Cobra group [Elapidae]
- Viper group [Viperidae]
- Sea snake group [Hydrophidae]

For many decades, the concept of the "Big 4" snakes of medical importance has reflected the view that 4 species responsible for Indian snakebite mortality. They are - Indian cobra (Naja naja),Common Krait (Bungarus caeruleus),Russell's viper (Daboia russelii) and Saw scaled viper (Echis carinatus).However, recently another species, the Hump-nosed pit viper (Hypnale hypnale), has been found to be capable of causing lethal envenomation, and this problem has not been recognised because of systematic misidentification of this species as the saw-scaled viper.

CIRCUMSTANCES OF SNAKE-BITES

Most snake bites happen when the snake is trodden on, either in the dark or in undergrowth, by someone who is bare footed or wearing only sandals. The snake may be picked up, unintentionally in a handful of foliage or intentionally by someone who is trying to show off. Some bites occur when the snake (usually a krait) comes into the home at night in search of its prey (other snakes, lizards, frogs, mice) and someone sleeping on the floor rolls over onto the snake in their sleep.[2]

EPIDEMIOLOGY

India: The numbers of snake-bite fatalities in India has long been controversial. Estimates as low as 61,507 bites and 1,124 deaths in 2006 and 76,948 bites and 1,359 deaths in 2007 and as high as 50 000 deaths each year have been published.[3] Previous studies including a field survey in randomly selected villages in Barddhaman (Burdwan) district, West Bengal suggested that among the total population of nearly five million people, nearly 8 000 were bitten and 800 killed by snakes each year, an average incidence of 16.4 deaths/100 000/year. In Maharashtra State, between 1974-78, there were an average of 1224 deaths/year (2.43 deaths/100 000/year). “The big four” medically important species had been

considered to be *Naja naja, Bungarus caeruleus*, *Daboia russelii* and *Echis carinatus* but other species have now been proved important in particular areas, such as *Naja oxiana*(north-west), N. kaouthia (north-east), *Hypnale hypnale* (south-west coast and Western Ghats (Joseph et al., 2007)), *Echis carinatussochureki*(Rajasthan) (Kochar et al., 2007) and *Trimeresurus malabaricus*(Hassan district, Mysore, Karnataka).

VENOM COMPOSITION

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

VENOM ENZYMES[4,5,6]

These include digestive hydrolases, hyaluronidase and activators or inactivators of physiological processes, such as kininogenase. Most venoms contain l-amino acid oxidase, phosphomono- and diesterases, 5'-nucleotidase, DNAase, NAD-nucleosidase, phospholipase A2 and peptidases.

Zinc metalloproteinase haemorrhagins: Damage vascular endothelium, causing bleeding.

Procoagulant enzymes: Venoms of Viperidae and some Elapidae and Colubridae contain serine proteases and other procoagulant enzymes that are thrombin-like or activate factor X, prothrombin and other clotting factors.

These enzymes stimulate blood clotting with formation of fibrin in the bloodstream. Paradoxically, this process results in incoagulable blood because most of the fibrin clot is broken down immediately by the body's own plasmin fibrinolytic system and, sometimes within 30 minutes of the bite, the levels of clotting factors are so depleted ("consumption coagulopathy") that the blood will not clot. Some venoms contain multiple anti-haemostatic factors.

Phospholipase A2 (lecithinase): The most widespread and extensively studied of all venom enzymes. It damages mitochondria, red blood cells,leucocytes, platelets, peripheral nerve endings, skeletal muscle, vascular endothelium, and other membranes, produces presynaptic neurotoxic activity,opiate-like sedative effects, leads to the autopharmacological release of histamine and anti-coagulation.

Acetylcholinesterase: Although found in most elapid venoms, it does not contribute to their neurotoxicity.

Hyaluronidase: Promotes the spread of venom through tissues.

Proteolytic enzymes (metalloproteinases, endopeptidases or hydrolases) and polypeptide cytotoxins (**"cardiotoxins"):** Increase vascular permeability causing oedema, blistering, bruising and necrosis at the site of the bite.

VENOM POLYPEPTIDE TOXINS ("NEUROTOXINS")

Postsynaptic (α) neurotoxins such as α-bungarotoxin and cobrotoxin, consist of 60-62 or 66-74 amino acids.They bind to acetylcholine receptors at the motor endplate. Presynaptic (β) neurotoxins such as β-bungarotoxin, crotoxin,and taipoxin, contain 120-140 amino acids and a phospholipase A subunit.

These release acetylcholine at the nerve endings at neuromuscular junctions and then damage the endings, preventing further release of transmitter.

CONSEQUENCES OF SNAKE-BITE

Victims of snake-bite may suffer any or all of the following:

1) Local envenoming confined to the part of the body that has been bitten. These effects may be debilitating, sometimes permanently.

2) Systemic envenoming involving organs and tissues away from the part of the body that has been bitten. These effects may be life threatening and debilitating, sometimes permanently.

3) Effects of anxiety prompted by the frightening experience of being bitten and by exaggerated beliefs about the potency and speed of action of snake venoms. These symptoms can be misleading for medical personnel.

4) Effects of first-aid and other pre-hospital treatments that may cause misleading clinical features. These may be debilitating and rarely even life-threatening.

Last two symptoms may develop in patients who are envenomed and in those who are not envenomed (bite by a non-venomous snake or by a venomous snake that failed to inject venom) or who were not in fact bitten by a snake at all but by a rodent or lizard or even impaled by a thorn.

SYMPTOMS AND SIGNS OF SNAKE-BITE

When venom has not been injected

Anxious people may hyperventilate so that they develop pins and needles sensation of the extremities, stiffness or tetany of their hands and feet and dizziness. Others may develop vasovagal shock

after the bite or suspected bite-faintness and collapse with profound slowing of the heart. Others may become highly agitated and irrational and may develop a wide range of misleading symptoms. Blood pressure and pulse rate may increase and there may be sweating and trembling. Another source of symptoms and signs not caused by snake venom is first aid and traditional treatments.[7] Constricting bands or tourniquets may cause pain, swelling and congestion that suggest local envenoming. Ingested herbal remedies may cause vomiting.

When venom has been injected

Early symptoms and signs

Following the immediate pain of mechanical penetration of the skin by the snake's fangs, there may be increasing local pain at the site of the bite, local swelling that gradually extends proximally up the bitten limb and painful enlargement of the regional lymph nodes draining the site of the bite.However, bites by kraits may be virtually painless and may cause negligible local swelling.If the biting species is unknown, the patient should be observed closely to allow recognition of the emerging pattern of symptoms, signs and laboratory results("the clinical syndrome"), together with other evidence, that may suggest which species was responsible.

Local symptoms and signs in the bitten part

1) fang marks

2) local pain

3) local bleeding

4) bruising,blistering

5) lymphangitis (raised red lines tracking up the bitten limb)

6) regional lymph node enlargement

7) inflammation (swelling, redness,warmth)

8) local infection, abscess formation necrosis

General

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

CARDIOVASCULAR MANIFESTATIONS(ELAPIDAE AND VIPERIDAE)

General symptoms of visual disturbances, dizziness, faintness, collapse, shock.Autonomic disturbances in the form of hypotension,hypertension,bradycardia,tachycardia,cardiac arrhythmias in the form of conduction blocks and ventricular

tachyarrythmias,myocardial involvement resulting in myocarditis,cardiomyopathy and acute pulmonary oedema.

Nayak et al have documented the range of cardiac manifestations in snake bite. The mechanism of cardiac involvement in neurotoxic snake bite is not clear. However it is possibly due to one of the myriad toxins seen in snake venom, which can cause morphological changes, enzyme alteration, ultrastructural disturbance and genetic alterations of the myocardial tissue.

BLEEDING AND CLOTTING DISORDERS (VIPERIDAE)

Traumatic bleeding from recent wounds (including prolonged bleeding from the fang marks and from old partly-healed wounds).

Spontaneous systemic bleeding - from gums,epistaxis, bleeding into the tears, intracranial haemorrhage (meningism from subarachnoid haemorrhage, lateralizing signs and/or coma from cerebral haemorrhage), haemoptysis, haematemesis, rectal bleeding or melaena, haematuria, vaginal bleeding, ante-partum haemorrhage in pregnant women, bleeding into the mucosae , skin (petechiae, purpura, discoid haemorrhages and ecchymoses) and retina.

Cerebral arterial thrombosis (Russell's v*iper Daboia russelii)* Thrombotic strokes, confirmed by angiography or imaging, are reported rarely after envenoming by *D. russelii*.[8]

NEUROLOGICAL MANIFESTATIONS (ELAPIDAE, RUSSELL'S VIPER)

Drowsiness, paraesthesiae, abnormalities of taste and smell, "heavy" eyelids, ptosis , external ophthalmoplegia, paralysis of facial muscles and other muscles innervated by the cranial nerves,bulbar paralysis resulting in nasal voice or aphonia,nasal regurgitation, difficulty in swallowing secretions, respiratory and generalised descending flaccid paralysis can all occur in Elapide and Russell's viper bites.

RHABDOMYOLYSIS (SEA SNAKES, SOME KRAIT SPECIES

Bungarus niger and B. candidus, **Russell's viper *Daboia russelii)***

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

RENAL MANIFESTATIONS(VIPERIDAE, SEA SNAKES)

Renal manifestations result in loin (lower back) pain, haematuria, haemoglobinuria, myoglobinuria, oliguria/anuria, symptoms and signs of uraemia (acidotic breathing, hiccups, nausea, pleuritic chest pain)[9] in Viperine and sea snake bites.

ENDOCRINE MANIFESTATIONS

Acute pituitary and adrenal insufficiency from infarction of the anterior pituitary (Russell's viper in Myanmar and South India)[10] has also been reported.This may manifest during the Acute phase as shock and hypoglycaemia.

In the chronic phase (months to years after the bite): Weakness, loss of secondary sexual hair, loss of libido, amenorrhoea, testicular atrophy and hypothyroidism may occur.

SYNDROMIC APPROACH TO SNAKE BITE

Syndrome-1

- Local envenoming (swelling etc.) with bleeding/clotting disturbances - **Viperidae (all species).**

Syndrome-2

- Local envenoming (swelling etc.) with bleeding/clotting disturbances, shock or acute kidney injury - **Russell's viper, Hump-nosed pit viper in Sri Lanka and South India.**
- Conjunctival oedema (chemosis) and acute pituitary insufficiency - **Russell's viper.**
- Ptosis, external ophthalmoplegia, facial paralysis etc and dark brown urine - **Russell's viper in Sri Lanka and South India.**

Syndrome-3

- Local envenoming (swelling etc.) with neuromuscular paralysis - **cobra or king cobra.**

Syndrome-4

Paralysis with minimal or no local envenoming.

- Bitten on land while sleeping on the ground – **krait.**
- Bitten in the sea, estuary and some freshwater lakes - **sea snake.**

Syndrome-5

Paralysis with dark brown urine and acute kidney injury

- Bitten on land (with bleeding/clotting disturbance) - **Russell's viper, Sri Lanka and South India.**
- Bitten on land while sleeping indoors - **krait *(B. niger, B. candidus, B. multicinctus).***
- Bitten in sea, estuary and some freshwater lakes (no bleeding/clotting disturbances) - **sea snake.**

LIMITATIONS OF SYNDROMIC APPROACH

The more carefully the clinical effects of snake-bites are studied, the more it is realized that the range of activities of a particular venom is very wide. For example, some elapid venoms, such as those of Asian cobras, can cause severe local envenoming, formerly thought to be an effect only of viper venoms. In Sri Lanka

and South India, Russell's viper venom causes paralytic signs (ptosis, etc.). Although there may be considerable overlap of clinical features caused by venoms of different species of snake, a "syndromic approach" may still be useful, especially in resource poor settings.

LONG TERM COMPLICATIONS OF SNAKE-BITE

At the site of the bite, loss of tissue may result from sloughing or surgical debridement of necrotic areas or amputation.Chronic ulceration, infection, osteomyelitis, contractures, arthrodesis or arthritis may persist causing severe physical disability . Malignant transformation may occur in skin ulcers after a number of years. Chronic kidney disease (renal failure) occurs after bilateral cortical necrosis (Russell's viper and hump-nosed pit viper bites) and chronic panhypopituitarism or diabetes insipidus after Russell's viper bites in Myanmar and South India.Chronic neurological deficit is seen in the few patients who survive intracranial haemorrhages and thrombosis (Viperidae).

MANAGEMENT OF SNAKE-BITE

- First aid treatment
- Transport to hospital
- Detailed clinical assessment
- Investigations/laboratory tests
- Antivenom treatment
- Observing the response to antivenom
- Deciding whether further dose(s) of antivenomare needed
- Treatment of the bitten part
- Rehabilitation
- Treatment of chronic complications

AIMS OF FIRST-AID

The main aims of first aid treatment are to retard the systemic absorption of venom and to preserve life and prevent complications before the patient can receive medical care and control distressing or dangerous early symptoms of envenoming.

The patient should be transported to a place where they can receive medical care.

RECOMMENDED FIRST-AID METHODS

Reassure the victim who may be very anxious.

Immobilize the whole of the patient's body by laying him/her down in a comfortable and safe position and especially, immobilize the bitten limb with a splint or sling. Any movement or muscular contraction increases absorption of venom into the blood stream and lymphatics.

If the necessary equipment and skills are available, consider pressure-immobilization or pressure pad unless an elapid bite can be excluded. Avoid any interference with the bite wound (incisions, rubbing, vigorous cleaning, massage, application of herbs or chemicals) as this may introduce infection, increase absorption of the venom and increase local bleeding.[11]

The use of tight tourniquets have been traditionally used to stop venom flow into the body following snakebite however there is an increased risk of ischemia and local necrosis and risk of massive neurotoxic blockade when tourniquet is released.

RELEASE OF TIGHT BANDS, BANDAGES AND LIGATURES

Ideally, these should not be released until the patient is under medical care in hospital, resuscitation facilities are available and antivenom treatment has been started .[12]

DETAILED CLINICAL ASSESSMENT AND SPECIES DIAGNOSIS

History

A precise history of the circumstances of the bite and the progression of local and systemic symptoms and signs is very important.

A common early symptom of systemic envenoming is vomiting.Patients who become defibrinogenated or thrombocytopenic may begin to bleed from old, partially-healed wounds as well as bleeding persistently from the fang marks. The patient should be asked how much urine has been passed since the bite and whether it was of a normal colour. Patients who complain of sleepiness, drooping eyelids or blurred or double vision may have neurotoxic envenoming.

An important early symptom of sea snake envenoming that may develop as soon as 30 minutes after the bite is generalized pain, tenderness and stiffness of muscles and trismus.

EARLY CLUES THAT A PATIENT HAS SEVERE ENVENOMING

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

PHYSICAL EXAMINATION

This should start with careful assessment of the site of the bite and signs of local envenoming.

EXAMINATION OF THE BITTEN PART

The extent of swelling, which is usually also the extent of tenderness to palpation (start proximally), should be recorded. Lymph nodes draining the limb should be palpated and overlying

ecchymoses and lymphangitic lines noted. A bitten limb may be tensely oedematous, cold, immobile and with impalpable arterial pulses.These appearances may suggest intravascular thrombosis, which is exceptionally rare after snake-bite, or a compartmental syndrome, which is uncommon. If possible, intracompartmental pressure should be measured and the blood flow and patency of arteries and veins assessed (e.g. by doppler ultrasound).Early signs of necrosis may include blistering, demarcated darkening (easily confused with bruising) or paleness of the skin, loss of sensation and a smell of putrefaction.

GENERAL EXAMINATION

Measure the blood pressure (sitting up and lying to detect a postural drop indicative of hypovolaemia) and heart rate.

Examine the skin and mucous membranes for evidence of petechiae, purpura, discoid haemorrhages), ecchymoses and, in the conjunctivae, for haemorrhages and chemosis.Thoroughly examine the gingival sulci, using a torch and tongue depressor, as these may show the earliest evidence of spontaneous systemic bleeding. Examine the nose for epistaxis.Abdominal tenderness may suggest gastrointestinal or retroperitoneal bleeding.Loin (low back) pain and tenderness suggests acute renal ischaemia (Russell's viper

bite). Intracranial haemorrhage is suggested by lateralising neurological signs, asymmetrical pupils, convulsions or impaired consciousness in the absence of respiratory or circulatory collapse. To exclude early neurotoxic envenoming, ask the patient to look up and observe whether the upper lids retract fully.Test eye movements for evidence of early external ophthalmoplegia. The muscles flexing the neck may be paralysed, giving the “broken neck sign”.

Cardiac manifestation is assessed by heart rate,blood pressure both supine and sitting,auscultation for gallop,murmurs and lung signs.

INVESTIGATIONS/LABORATORY TESTS

20-MINUTE WHOLE BLOOD CLOTTING TEST (20WBCT)

Place 2 ml of freshly sampled venous blood in a small, new or heat cleaned, dry, glass vessel. Leave undisturbed for 20 minutes at ambient temperature. Tip the vessel once. If the blood is still liquid (unclotted) and runs out, the patient has hypofibrinogenaemia (“incoagulable blood”) as a result of venom-induced consumption coagulopathy. In the South-East Asia region, incoagulable blood is diagnostic of a viper bite and rules out an elapid bite.

Haemoglobin concentration / Haematocrit: A transient increase indicates haemo concentration resulting from a generalized increase in capillary permeability (e.g. in Russell's viper bite).Decrease reflects blood loss or intravascular haemolysis.

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

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

Blood film:Fragmented red cells ("helmet cell", schistocytes) are seen whenthere is microangiopathichaemolysis.

Biochemical abnormalities: Aminotransferases and muscle enzymes (creatine kinase, aldolase etc) will be elevated if there is severe local muscle damage or particularly, if there is generalized muscle damage (sea snake, some krait, Sri Lankan and South Indian Russell's viper bites).

Mild hepatic dysfunction is reflected in slight increases in other serum enzymes. Bilirubin is elevated following massive extravasation of blood. Potassium, creatinine, urea or blood urea nitrogen levels are raised in the renal failure of Russell's viper, hump-nosed viper bites and sea snakebites.

Early hyperkalaemia may be seen following extensive rhabdomyolysis in sea snake-bites. Bicarbonate will be low in metabolic acidosis (e.g. renal failure).

ABG and pH: may show evidence of respiratory failure (neurotoxic envenoming) and acidaemia (respiratory or metabolic acidosis).

Urine examination: The colour of the urine (pink, red, brown, black) should be noted and the urine should be tested by dipsticks for blood or haemoglobin or myoglobin.Red cell casts indicate glomerular bleeding.Massive proteinuria is an early sign of the generalized increase in capillary permeability in Russell's viper envenoming and an early indicator of acute kidney injury

ECG AND ECHO: To assess the cardiac rhythm and contractile function respectively.

ANTI SNAKE VENOM (ASV)

Anti snake venom (ASV) in India is polyvalent i.e. it is effective against all the four common species; Russells viper (*Daboia russelii*), Common Cobra (*Naja naja*), Common Krait (*Bungarus caeruleus*) and Saw Scaled viper (*Echis carinatus*). There are no currently available monovalent ASVs primarily because there are no objective means of identifying the snake species,in the absence of dead snake.

ASV ADMINISTRATION CRITERIA[13]

ASV is a scarce, costly commodity and should only be administered when there are definite signs of envenomation. Unbound free flowing venom can only be neutralized when it is in the blood stream or tissue fluid.In addition ASV carries the risk of anaphylactic reactions and should not therefore be used unnecessarily.

ASV should be administered ONLY if a patient develops one or more of following signs and symptoms of envenomation.

SYSTEMIC ENVENOMING

- Evidence of coagulopathy: Primarily detected by 20 WBCT or visible spontaneous systemic bleeding, gums etc.

- Evidence of neurotoxicity: ptosis, external ophthalmoplegia, inability to lift the head etc.

The above two methods of establishing systemic envenomation are the primary determinants.In the Indian context one of these two categories will be the sole determinant of whether ASV is administered to a patient or not.The other indications for ASV are

- Cardiovascular abnormalities: Hypotension, shock, cardiac arrhythmia, abnormal ECG.
- Acute kidney injury: oliguria/anuria, rising blood creatinine/ urea.
- Haemoglobin /myoglobinuria:dark brown urine,urine dipsticks, other evidence of intravascular haemolysis or generalised rhabdomyolysis.
- Persistent and severe vomiting or abdominal pain.

CURRENT LOCAL ENVENOMING

- Severe current, local swelling involving more than half of the bitten limb (in the absence of a tourniquet). In the case of severe swelling after bites on the digits.

- Rapid extension of swelling(for example beyond the wrist or ankle within a few hours of bite on the hands or feet).

- Development of an enlarged tender lymph node draining the bitten limb.

NO ASV TEST DOSE MUST BE ADMINISTERED

Test doses have been shown to have no predictive value in detecting anaphlactoid or late serum reactions.

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

Neurotoxic/ Anti Haemostatic (8-10 vials)

All ASV should be administered over 1 hour period. The patient should be closely monitored for 2 hours.

NEUROTOXIC ENVENOMATION

Neostigmine is an anticholinesterase that prolongs the life of acetylcholine and can therefore reverse respiratory failure and neurotoxic symptoms. It is particularly effective for post synaptic neurotoxins such as those of the Cobra.[14,15] There is some doubt over its usefulness agaist presynaptic neurotoxin such as those of the Krait and Rusell viper.[16]

In the case of neurotoxic envenomation the Neostigmine test will be administered with a dose of 1.5 mg of neostigmine i.m along with 0.6 mg of Atropine i.v.

The patient should be closely monitored for 1 hr to determine if neostigmine is effective.

The followingare useful objective methods to assess this:

1) Single breath count

2) Mm of Iris uncovered (Amount covered by the descending eyelid)

3) Inter incisor distance

4) Length of time upward gaze can be maintained

5) FEV 1 or FVC (If available)

REPEAT DOSES: ANTI HAEMOSTATIC

In the case of anti haemostatic envenomation, the ASV strategy is based on a 6 hour time period.When the initial test is abnormal initial ASV dose will be given over 1 hour.

After 6 hours a further coagulation test should be performed and a further dose should be administered in the event of continued coagulation disturbance.

This is due to inability of liver to replace clotting factors within 6 hours. WBCT and ASV dosing should be done on 6 hourly basis until coagulation is restored.

The repeat dose should be 5-10 vials of ASV i.e. half to one full dose of the original dose.

REPEAT DOSES: NEUROTOXIC

The ASV regime relating to neurotoxic envenomation has caused considerable confusion. If the initial dose has been unsuccessful in reducing the symptoms or if the symptom have worsened or if the patient has gone into respiratory failure then a further dose should be administered, after 1-2 hours. At this point the patient should be re-assessed. If the symptoms have not improved or worsened second dose of ASV can be given. This dose should be the same as the initial dose, i.e. if 10 vials were given initially then 10 vials should be repeated for a second dose and then ASV is discontinued. 20 vials is the maximum dose that should be given to neurotoxically envenomed patient.

HYPOTENSION AND PULMONARY EDEMA

Hypotension can have a number of causes, particularly loss of circulating volume due to haemorrhage, vasodilation due to the action of the venom or direct effects on the heart. Treatment is by means of plasma expanders.Pulmonary edema is managed by diuretics and ventilator support.

ANTIVENOM REACTIONS:

ASV reactions manifest as

(1)Early anaphylactic reactions,

(2)Pyrogenic reactions,

(3)Late Serum sickness like reactions.

ASV should be stopped at the earliest sign of a reaction.

Epinephrine(0.1% solution) is the effective treatment for early anaphylactic and pyrogenic antivenom reactions.In patients with early anaphylactic and pyrogenic antivenom reactions: Epinephrine is given intramuscularly (into upper lateral thigh) in an initial dose of 0.5 mg for adults and 0.01 mg/kg body weight for children.

Late serum sickness like reactions are treated with oral antihistamines,if refractory with oral steroids for 5-7 days.

MATERIALS AND METHODS

MATERIALS

All cases of Snake Bite coming to the hospital will be admitted and form the subjects of the study.

DESIGN OF STUDY

Prospective study

Sample size : 100

Human study or not : Yes, it involves Humans only

Ethical clearance status: Obtained

Period Of Study:7 Months

PLACE OF STUDY

Poison Control, Training& Research Centre, Rajiv Gandhi Government General Hospital, Madras Medical College, Chennai-3.

METHODS

1) Inclusion criteria:

- All admitted cases of snake bite will be evaluated to assess poisonous status.

2) Exclusion criteria:

- Those with existing cardiac disease, children, pregnant women, immune suppressed persons, those with any other chronic illness will be excluded from the study.

3) Informed consent will be obtained from each case included for the study.

4) Poisonous cases will be grouped according to Elapidae, Viperidae or other bites by syndromic approach.

5) Snakes will be identified when it is brought by the victim/relatives.

6) Details will be documented in a proforma (enclosed).

7) Laboratory investigations will be done as in proforma(enclosed).

8) Additional biochemical parameters likely to influence the cardiac function.

9) Cardiac screening by ECG and ECHO will be carried at Poison control, Training & Research Centre, GGH, Chennai. ECG, ECHO will be done on DAY 1, 3 and repeated whenever required.

10) Patients will be followed up daily while in the hospital.

11) Patients will be provided appropriate treatment.

12) Follow up :Cases with cardiac involvement will be followed up after 4 weeks by ECG and ECHO.

13) Data will be entered into the excel sheet of the computer.

14) Data will be analysed using SPSS package.

RESULTS AND OBSERVATIONS

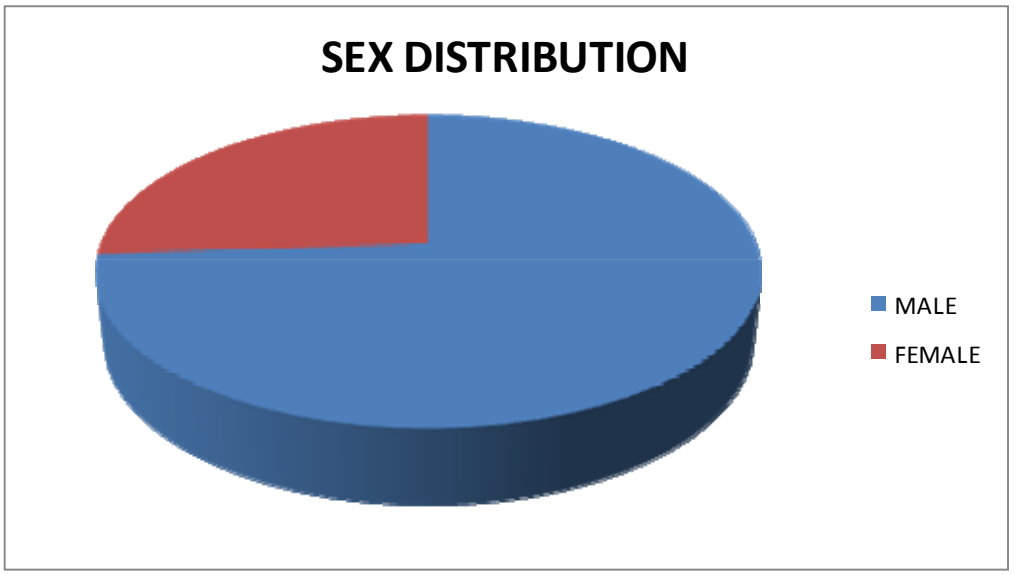


TABLE 1:

SEX	PERCENTAGE (%)
MALE	74
FEMALE	26

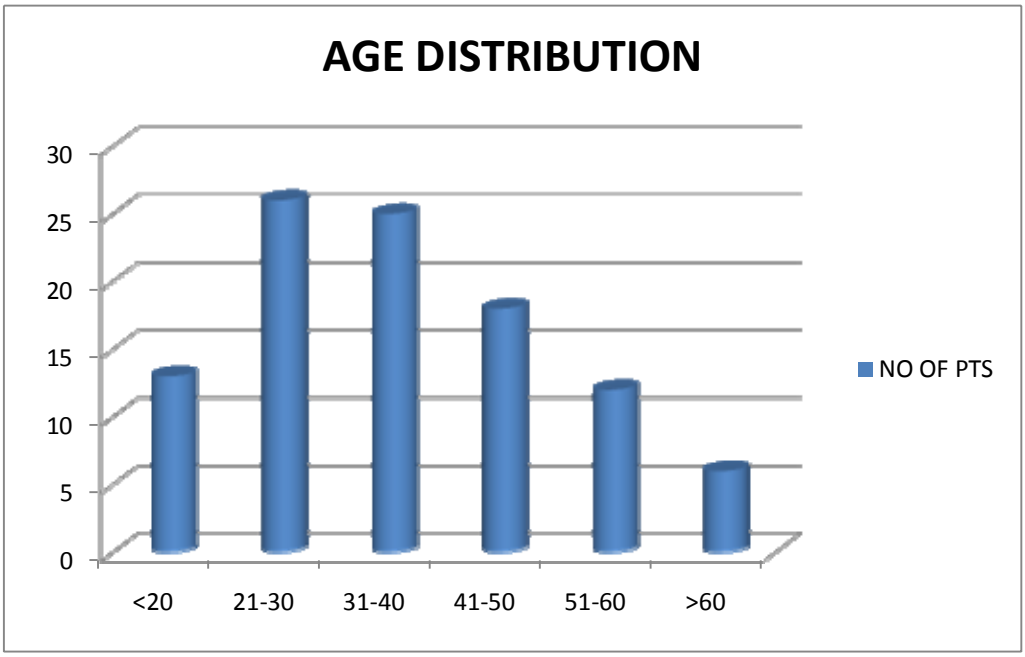


TABLE 2:

AGE GROUP (IN YEARS)	PERCENTAGE (%)
< 20	13
21 - 30	26
31 – 40	25
41 – 50	18
51 – 60	12
> 60	6

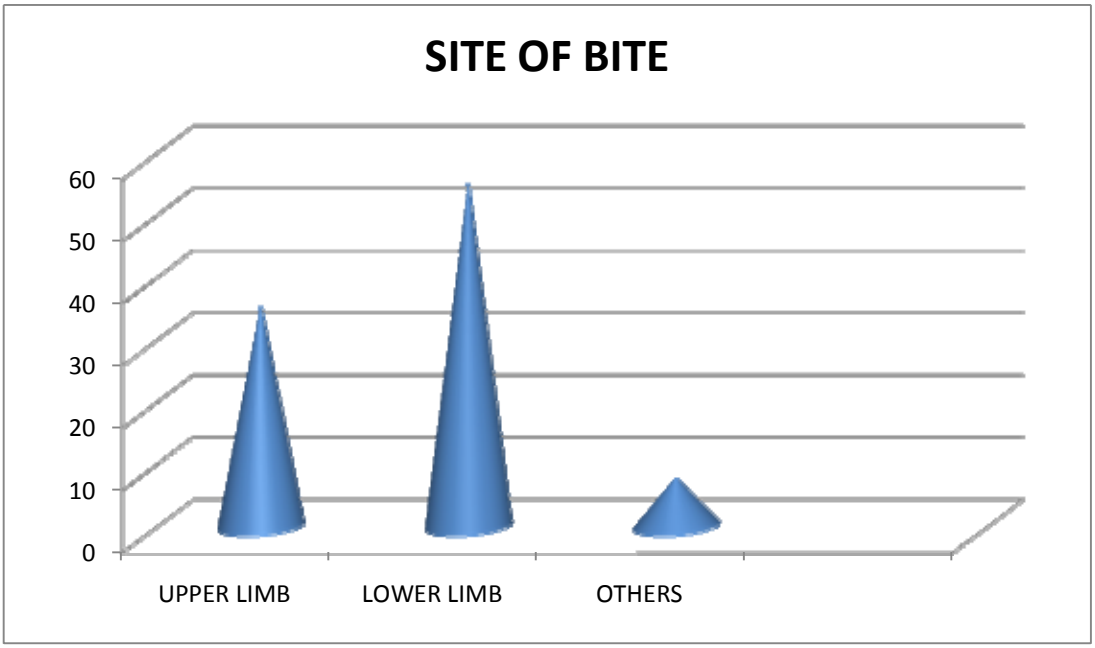


TABLE 3:

SITE OF BITE	PERCENTAGE (%)
UPPER LIMB	36
LOWER LIMB	56
OTHERS	8

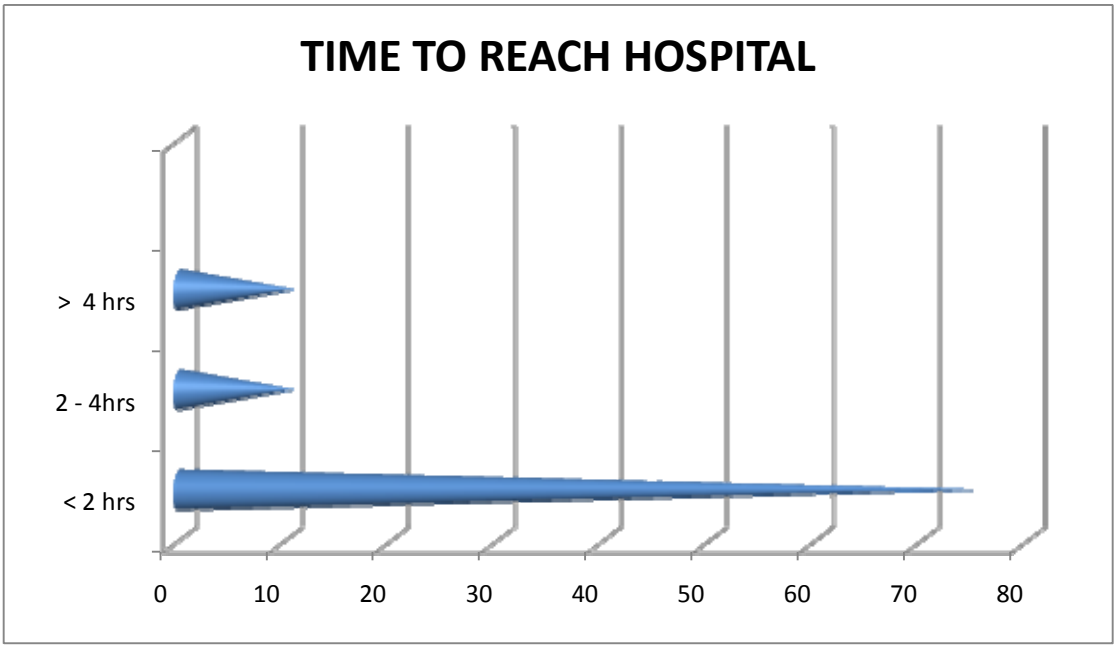


TABLE 4:

TIME TO REACH HOSPITAL	PERCENTAGE (%)
< 2 HOURS	78
2 – 4 HOURS	11
>4 HOURS	11

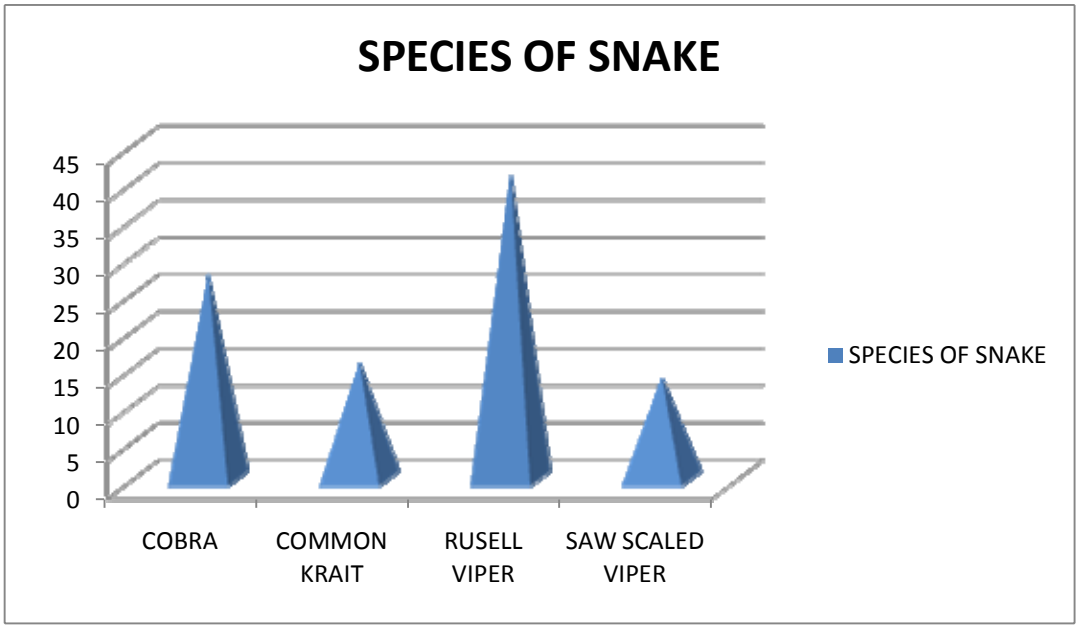


TABLE 5:

SPECIES OF SNAKE	PERCENTAGE
COBRA	28
COMMON KRAIT	16
RUSELL VIPER	42
SAW SCALE VIPER	14

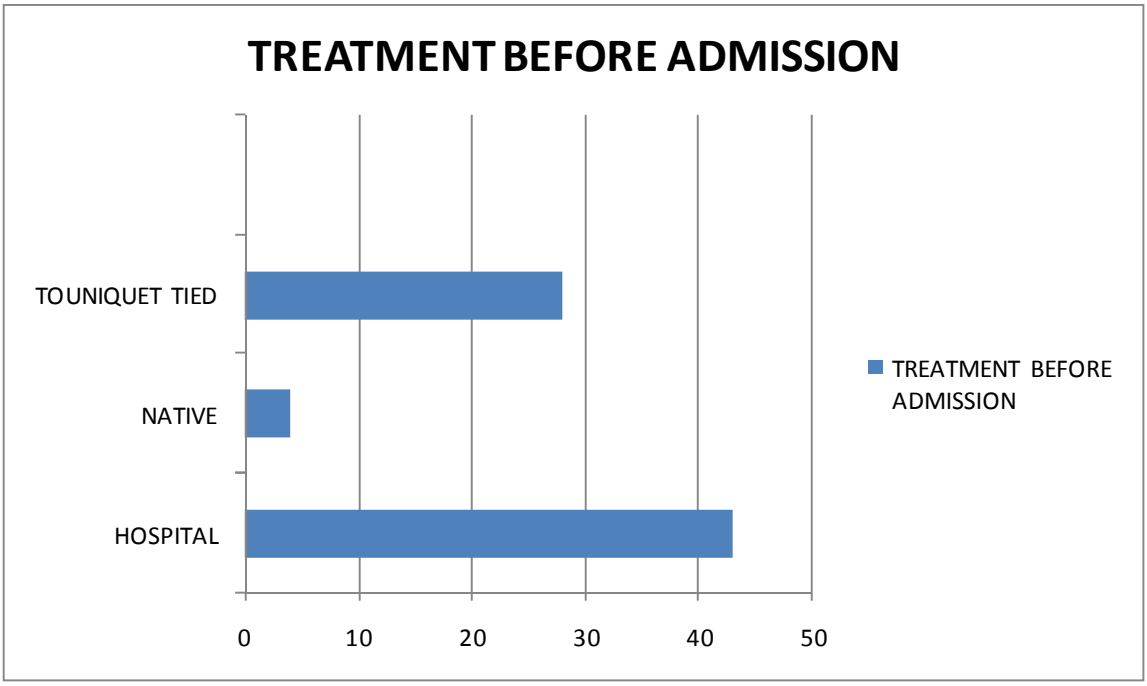


TABLE 6:

TREATMENT BEFORE ADMISSION	PERCENTAGE (%)
HOSPITAL	43
NATIVE	04
TOURNIQUET TIED	28

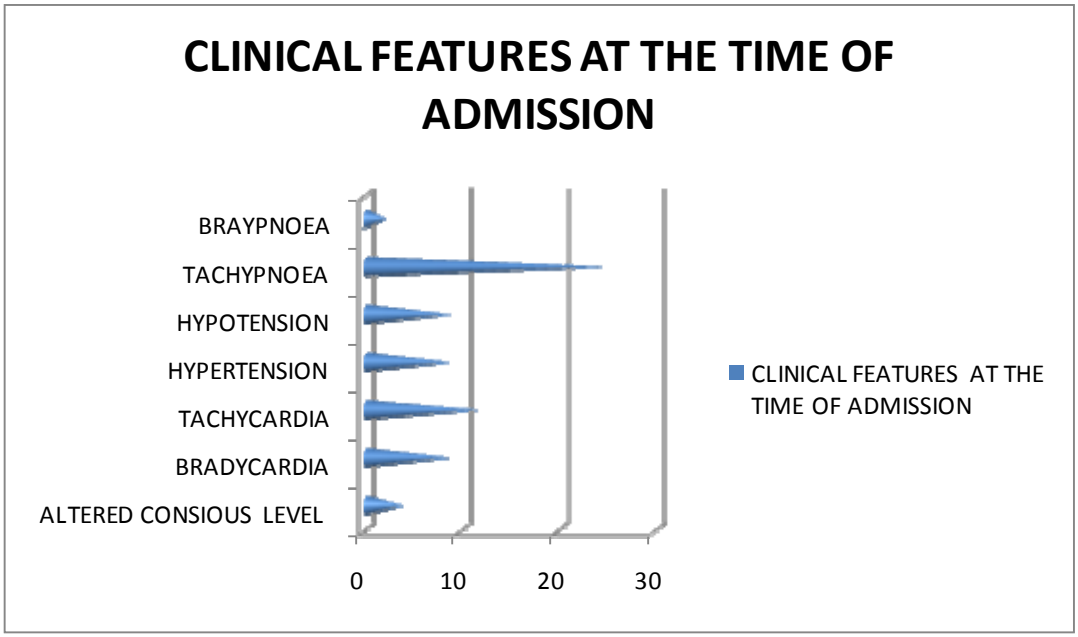


TABLE 7:

CLINICAL FEATURES ON ADMISSION	FREQUENCY
ALTERED CONSIOUS LEVEL	04
BRADYCARDIA	09
TACHYCARDIA	12
HYPOTENSION	09
HYPERTENSION	09
TACHYPNOEA	26
BRADYPNOEA	02

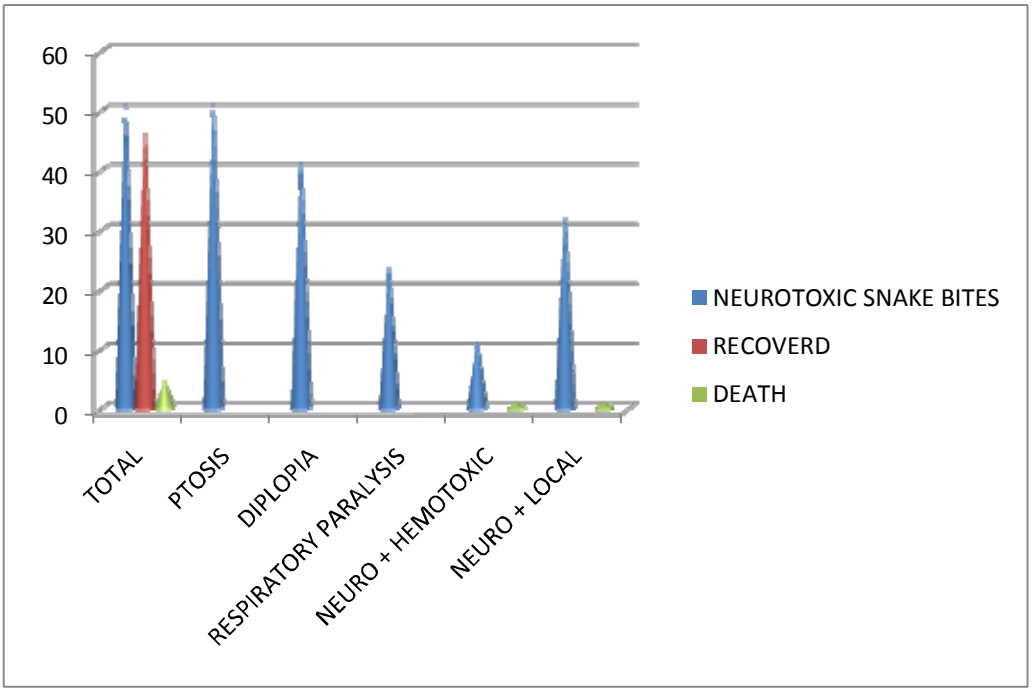


TABLE 8: NEUROTOXICITY

MANIFESTATIONS	PERCENTAGE (%)
PTOSIS	55
DIPLOPIA	45
RESPIRATORY PARALYSIS	25
NEUROTOXICITY+HEMOTOXICITY	12
NEUROTOXICITY + LOCAL ENVENOMATION	35

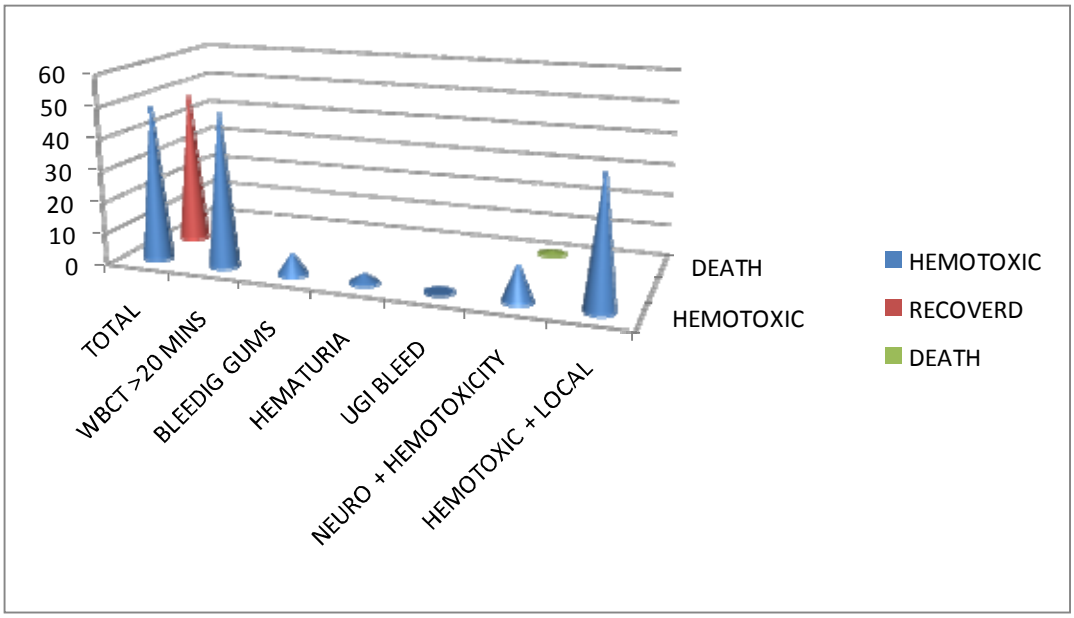


TABLE 9: HEMOTOXICITY

MANIFESTATIONS	PERCENTAGE (%)
WBCT > 20 MINS	52
BLEEDING GUMS	7
HEMATURIA	3
UGI BLEED	0
NEUROTOXICITY+HEMOTOXICITY	12
HEMOTOXICITY+LOCAL	43

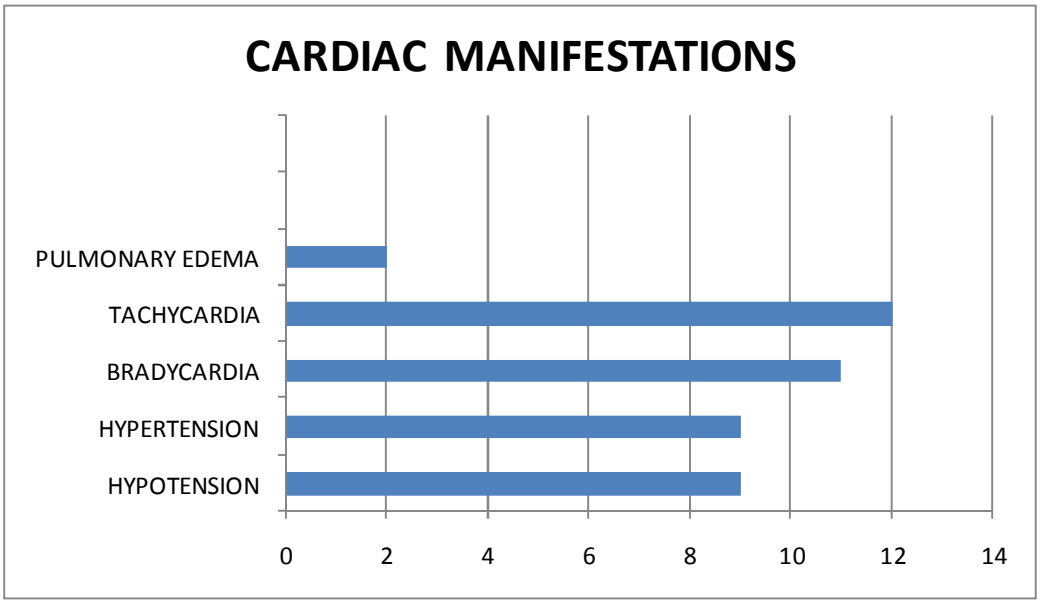


TABLE 10:

CARDIAC MANIFESTATIONS	FREQUENCY
HYPOTENSION	09
HYPERTENSION	09
BRADYCARDIA	11
TACHYCARDIA	12
PULMONARY EDEMA	02

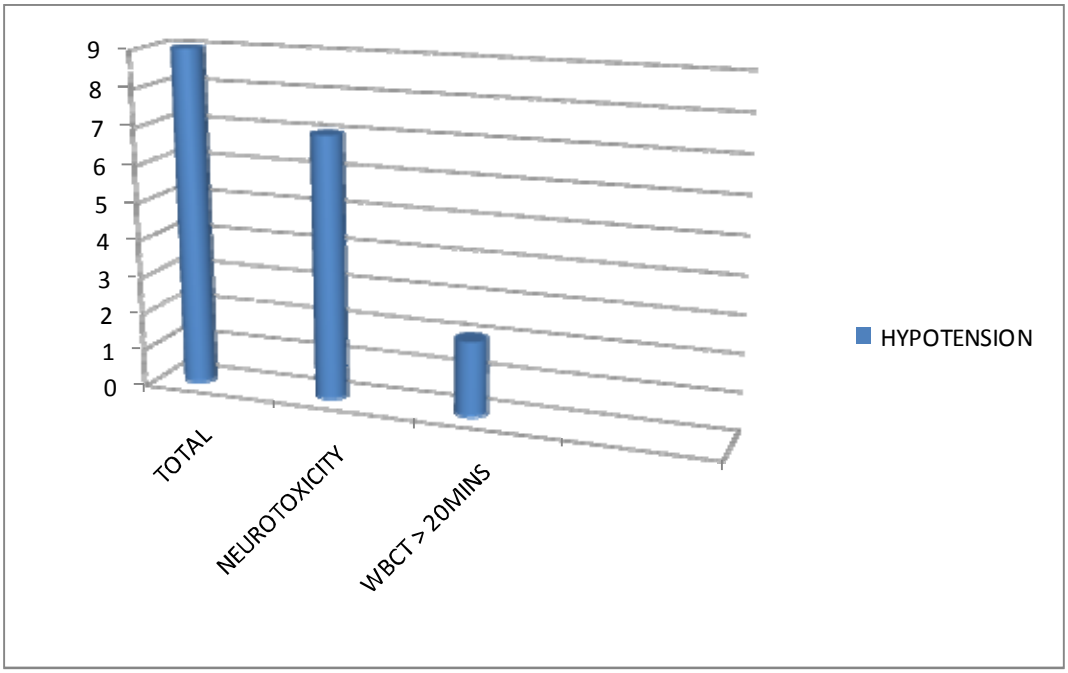


TABLE 11: CARDIAC MANIFESTATION IN THE FORM OF HYPOTENSION

MANIFESTATION	PERCENTAGE
NEUROTOXICITY	7
WBCT > 20 MINS	2
TOTAL	9

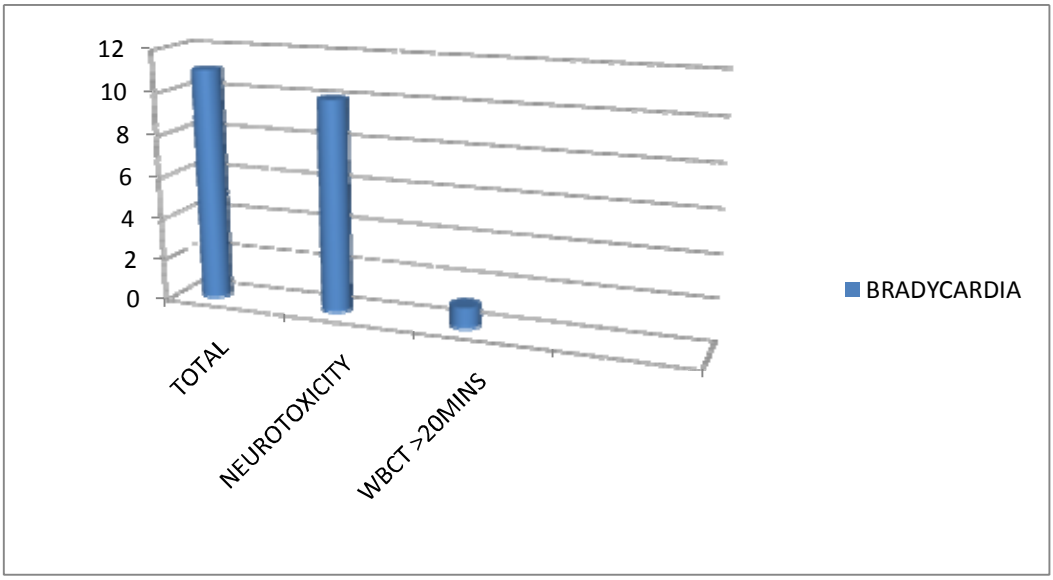
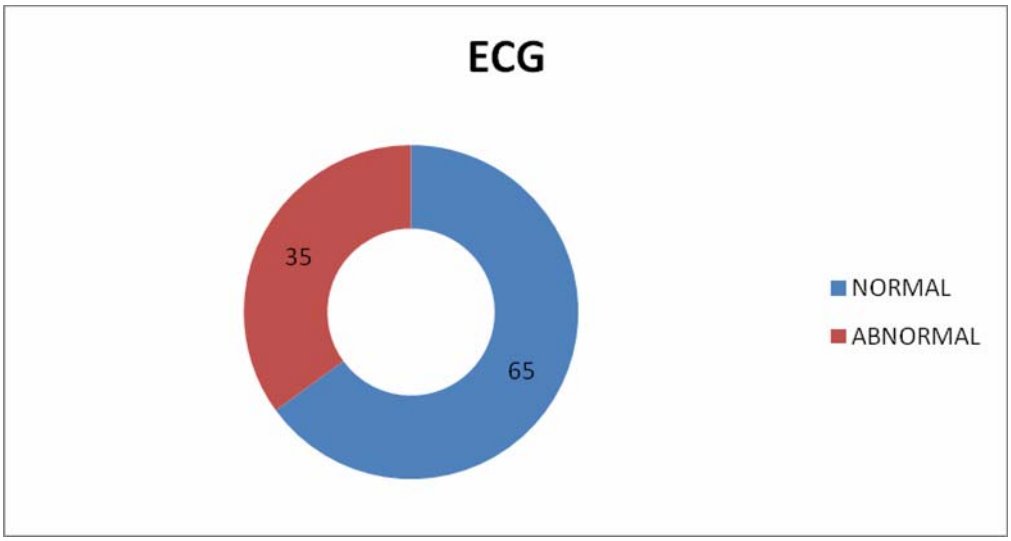


TABLE 12: CARDIAC INVOVEMENT IN THE FORM OF BRADYCARDIA

MANIFESTATION	PERCENTAGE
NEUROTOXICITY	10
WBCT >20 MINS	1
TOTAL	11



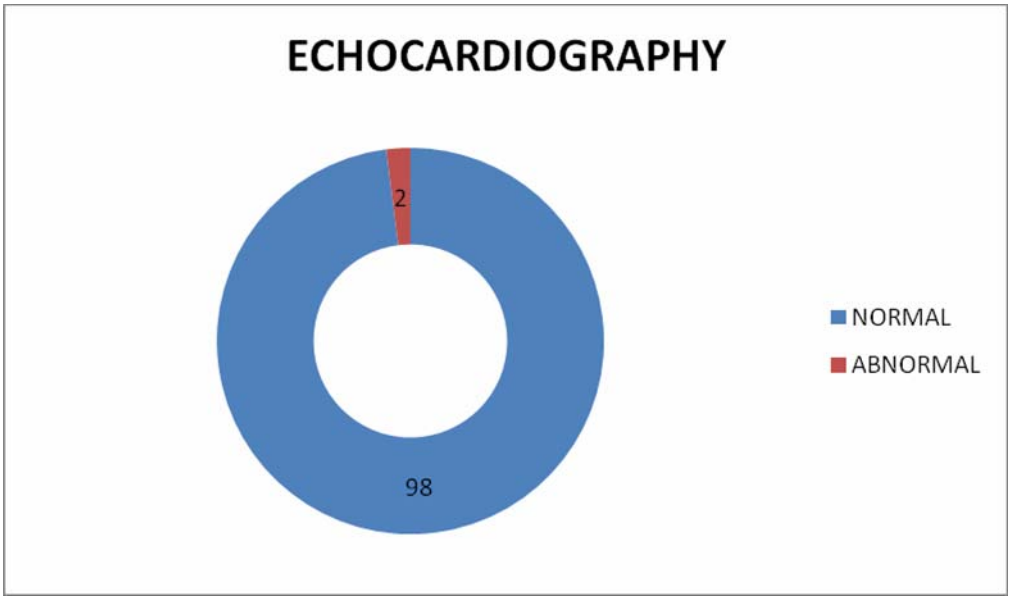


TABLE 13:

	NORMAL	ABNORMAL
ECG	65	35
ECHO	98	02

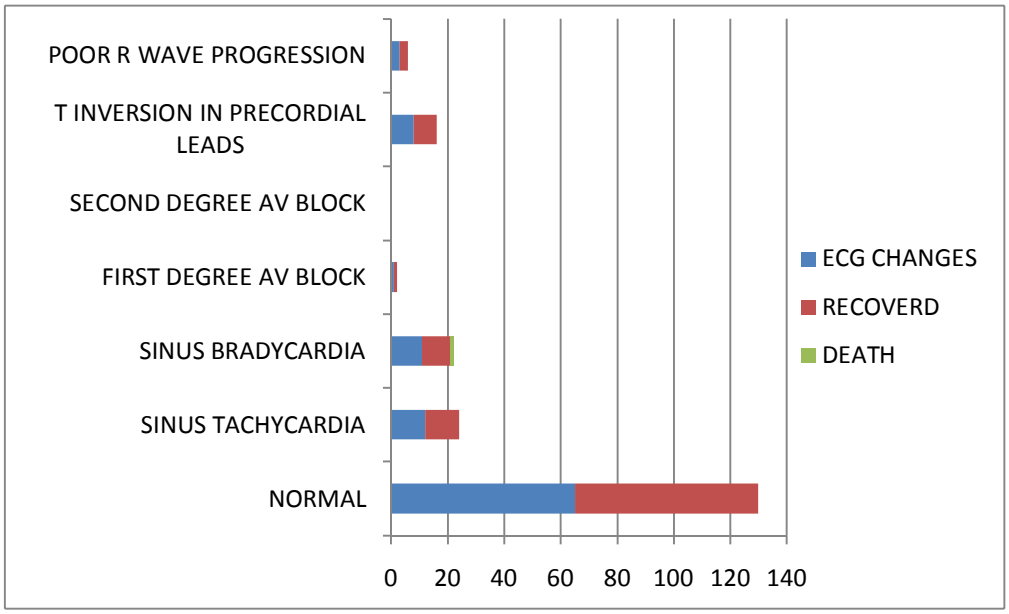


TABLE 14:

ECG CHANGES	PERCENTAGE (%)	RECOVERED	DEATH
NORMAL	65	65	
SINUS TACHYCARDIA	12	12	
SINUS BRADYCARDIA	11	10	1
FIRST DEGREE AV BLOCK	1	1	
SECOND DEGREE AV BLOCK	0	0	
T INVERSION IN PRECORDIAL LEADS	8	8	
POOR R WAVE PROGRESSION IN PRECORDIAL LEADS	3	3	

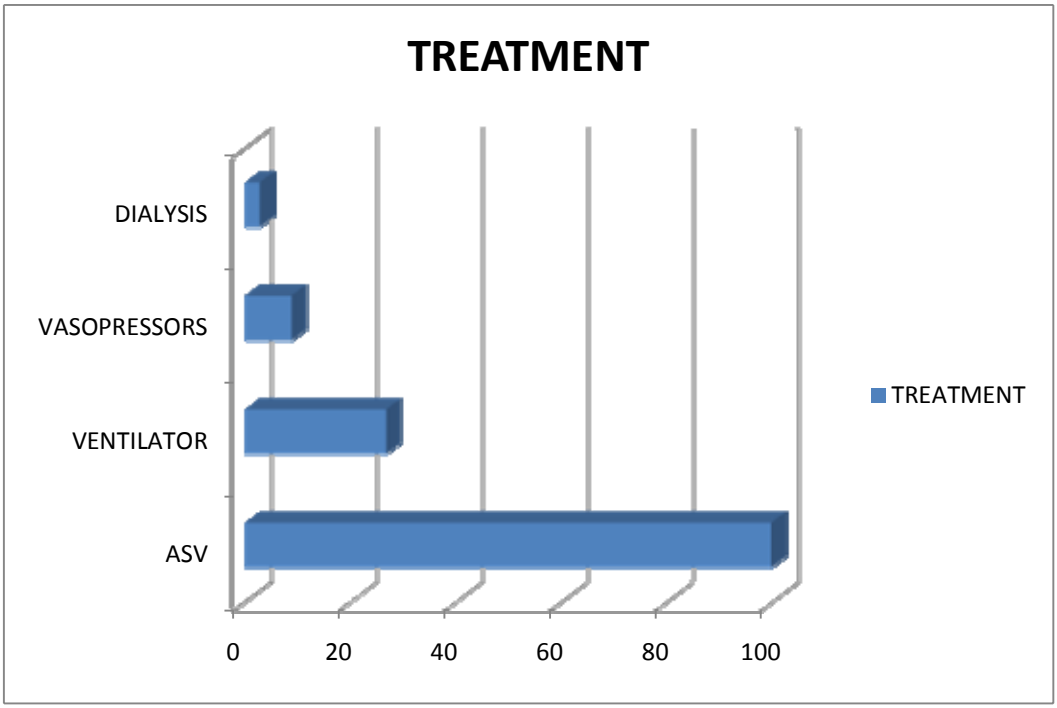
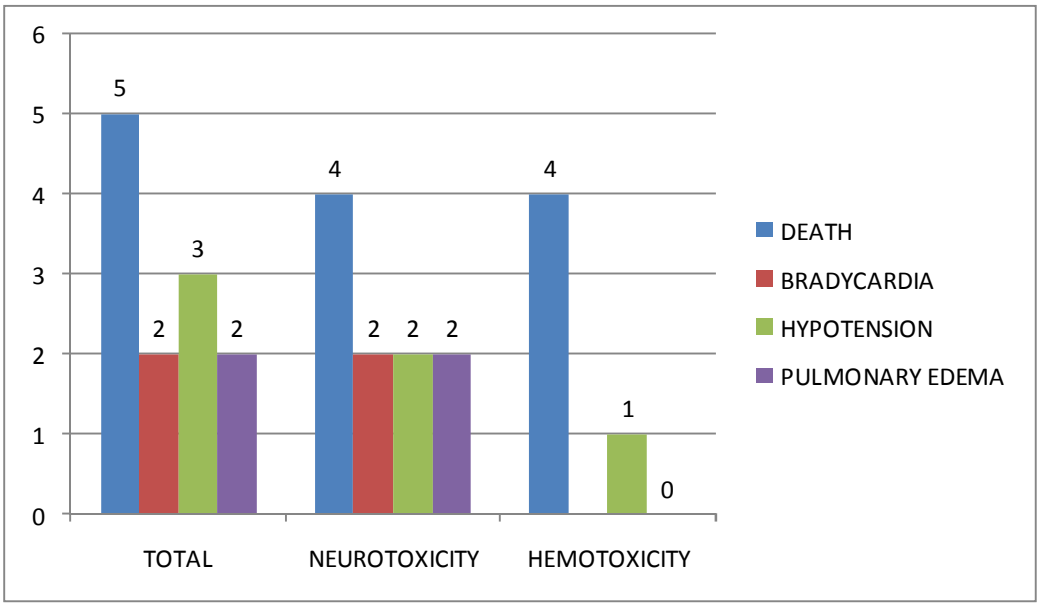


TABLE 15:

TREATMENT	PERCENTAGE
ASV	100
MECHANICAL VENTILATION	27
VASOPRESSORS	9
DIALYSIS	3

MORTALITY:


6
5
4
3
2
1
0
5
2
3
2
4
2
2
2
4
1
0
TOTAL
NEUROTOXICITY
HEMOTOXICITY
DEATH
BRADYCARDIA
HYPOTENSION
PULMONARY EDEMA

RESULTS

The present study observed that 26 per cent of patients of snake bite were in 2nd and 3rd decades of life,74% males and 26% females,56%of bites occurred in the lower extremities and 78 % of victims reached a healthcare facility in less than 2 hours.Viperine snake bite occurred in 56 per cent and Elapide snake bite occurred in 44 per cent of cases.

Cardiac involvement manifested in the form of alterations in the heart rate,blood pressure and electrocardiographic changes.

The disturbance in heart rate was seen in 23% per cent.Out of the 100 patients tachycardia was present in 12 per cent and bradycardia in 11 per cent of cases.

Hypertension was found in 9 per cent, hypotension in 9 per cent.Two patients had evidence of pulmonary edema.

Electrocardiography revealed changes in the form of sinus tachycardia(12%),sinus bradycardia (11%),myocardial ischemia (8%), and Poor R wave progression (3%).

One patient had electrocardiographic features of global T wave inversion and echocardiographic features of apical

hypokinesia suggestive of cardiomyopathy which reversed after a month.

The mortality rate was 5 per centof which 4 patients had neurotoxic features,1 patient had hemotoxic features and other 2 had evidence of pulmonary edema with abnormal electrocardiographic changes.

Postmortem was conducted in one patient.Section from heart showed normal cardiac muscle fibrosis with intervening interstium showing areas of hemorrhage. No evidence of coagulation necrosis is seen. Section from aorta shows thickened media and adventitia showing congested blood vessels.

DISCUSSION

The present study was conducted in 100 cases of snake bite to understand the pattern of cardiac involvement and to render help in the management of the problems arising out of them.All patients were subjected to routine clinical examination and specific investigations.

EPIDEMIOLOGY OF SNAKE BITE

Age and Sex Distribution:

In this series of 100 cases ,the number of the patients below 20 years of age were(13%), between 21-30 were (26%) ,between 31-40 were (25%) ,between 41-50 were (18%) ,above 50 (18%)).In this series males constituted about 74% and females about 26%. In study by Nayak et al,[17] 57% patients were in the 2nd and 3rd decade of life. The series reported in this study had the similar pattern of age group affection. This young age group affected by exposure forms the viable entity of any population both in terms of procurement and productivity. This case study and the case reports mentioned above throw light on the target age group

for educative and preventive programs to reduce the incidence of snake bite.

SITE OF BITE AND SPECIES OF SNAKE:

In this series 36% of bites occurred in the upper extremities, 56% of bites occurred in the lower extremities,8% of bites occurred in other areas such as head, trunk etc. Most bite in areas other than extremities occurred in Krait bites. Among 100 patients species identification was done depending upon the dead snake brought by the victims or by clinical manifestations using the syndromic approach. Cobra bite occurred in 28%, Common krait bite occurred in 16%, Russell viper bite occurred in 42%, Saw scaled viper bite occurred in 14%. In study by Nayak et al [17] Viperine snake bites occurred in 93 per cent and elapide snake bite in 7 per cent of cases.

TIME TO REACH HOSPITAL AND TREATMENT BEFORE ADMISSION

In this series 78% of victims arrived at hospital within 2 hours, 11% arrived between 2-4 hours and 11% reached after 4 hours. In these 100 cases 43 cases were treated and referred from nearby GH and PHC's belonging to five districts (Chennai, Thiruvallur, Kancheepuram, Villupuram and Vellore).4% of patients had native treatment and 28% had tourniquet tied above

the site of bite, which may be complicating factor for local envenomation which was present in 78% of patients in this study. Tourniquet use is contraindicated for use in India for the following reasons because of risk of ischemia and loss of the limb, increased risk of necrosis with 4/5 of the medically significant snakes of India, increased risk of massive neurotoxic blockade when tourniquet is release, risk of embolism if used in viper bites.

In addition,there is a danger of hypotension due to vasodilatory effects of venom when the tourniquet is released.They give patients a false sense of security, which encourages them to delay their journey to hospital. In the study by Nayaket al[17] seventy-six per cent of the patients presented within 24 hours of the bite.

CLINICAL FEATURES AT THE TIME OF ADMISSION:

Among 100patient 4% presented with altered sensorium,12% had tachycardia,9% had bradycardia.Hypertension[24] was present in 9% and hypotension in 9%.26% of patients were tachypnoeic and 9 % of patients were bradypnoeic. In the study by Nayak et al[17] tachycardia was found in 36.7 per cent and bradycardia in 10 per cent cases.

Hypertension was found in 6.7 per cent, hypotension in 16.7 per cent. Thirty per cent of patients had gallop rhythm. In our study two patients had evidence of pulmonary edema [41] with bilateral end inspiratory fine crepitations in both lung bases and no patient in our study had gallop rhythm.

TOXIC MANIFESTATIONS

Neurotoxicity was present in 55%,neurotoxicity and hemotoxicity combined in 12% and neurotoxicity and local envenomation combined in 35%.Hemotoxicity was present in 52% of patients in the form of prolonged WBCT >20 Mins. Other bleeding manifestations like bleeding gums and hematuria were present in 7% and 3% of patients respectively.Combined hemotoxicity and local envenomation were present in 43% of patients. The higher percentage of combination of neurotoxic, hemotoxic and local envenomation was due to the higher percentage of Rusell viper bites in this series.

CARDIOVASCULAR MANIFESTATIONS

Among 9% of patients with hypotension neurotoxicity was present in 7% and hemotoxicity was present in 2%.Among 11% of patients with bradycardia 11% had neurotoxic manifestations and1% had both neuro and hemotoxic manifestations. In this series

of 100,2 patients presented with features of acute pulmonary edema.

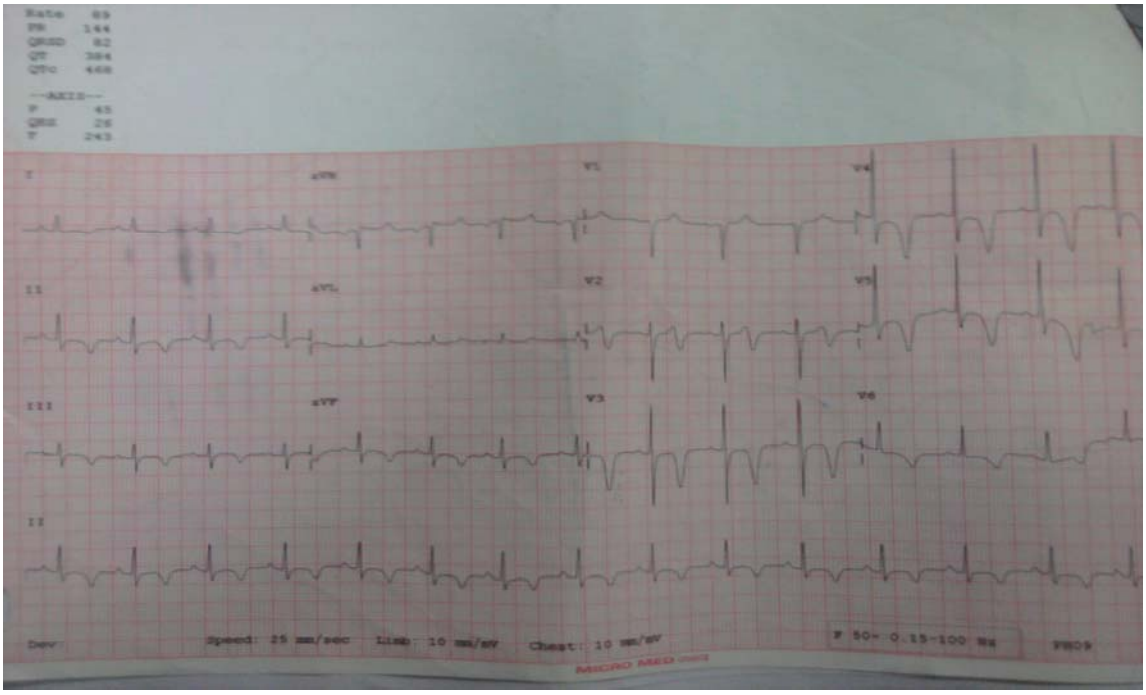
Pulmonary edema in snake bite may be secondary to myocarditis,[35,36] non cardiogenic pulmonary oedema secondary to the ASV used.[26] However, thisphenomenon generally occurs within six hours after Antivenin administration. The use of cardiodepressive drugs, e.g. benzodiazepine for sedation while performing bronchoscopy can also trigger pulmonary oedema. Other contributory causes, e.g. ischaemic insult to the heart secondary to hypoxia from delayed intubation, aspiration/mucous plugging with poor ventilation, can contribute to the causation of pulmonary oedema in patients with snake bites. The mechanism of cardiac involvement in neurotoxic snake bites is not clear[18] but is likely to be due to one of the myriad toxins seen in snake venom, which can cause morphological changes, enzyme alterations, ultra structural disturbances and genetic alterations of the myocardial tissue. In a recent experimental study, analysis of gene expression profiles in mice in response to cobra venom treatment revealed 203 genes in the heart, brain, kidney, liver and lung whose expressions were altered by at least three-fold.[19,21] Of these, 50% were

differentially expressed in the heart, and included genes involved in inflammation, apoptosis, ion transport and energy metabolism.[25,28]

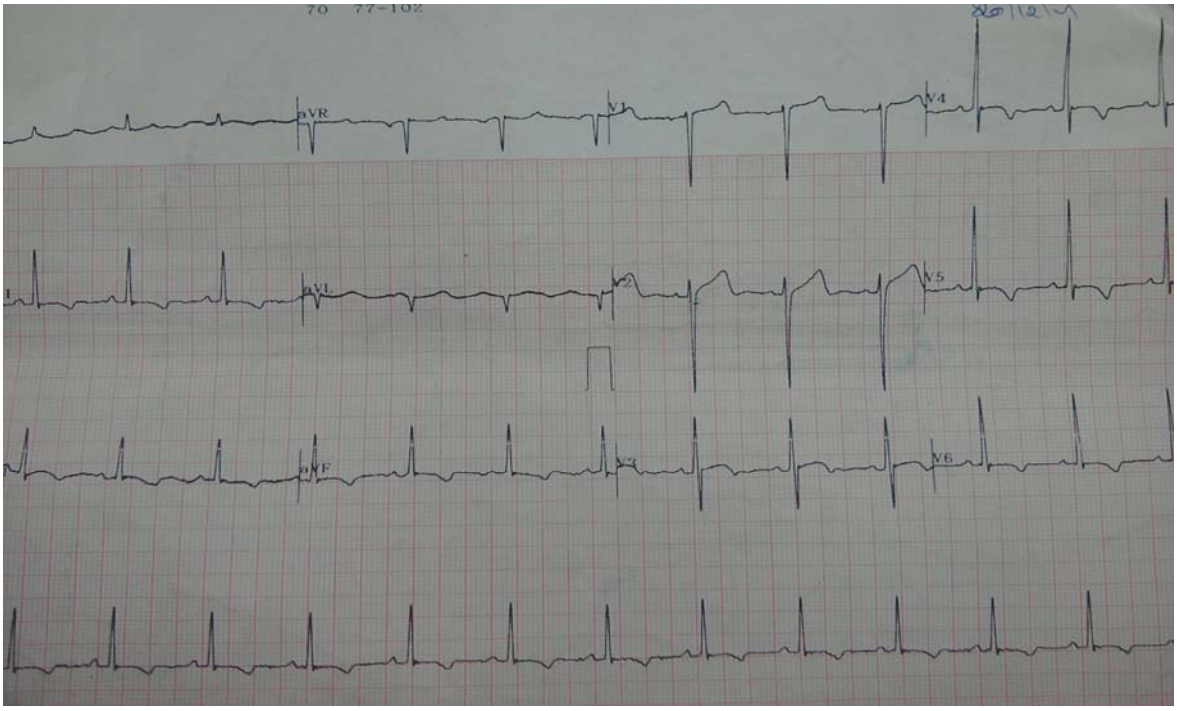
ECG FEATURES:

Among the 100 patients, the significant ECG abnormalities were found in 35%, 65% had normal rate and rhythm.Of the 35%,11% had Sinus Bradycardia, 12% had Sinus Tachycardia,1% had First degree Heart block. T wave inversion in precordial leads was present in 8% and Poor R wave progression in precordial leads was present in 3% of patients. The ECG changes were transient and recovered after treatment of snake bite. Ventricular ectopics and Bundle branch blocks were not found in our study[38,39] .In study by Nayaketal[17]electrocardiographic changes were sinus tachycardia, sinus arrhythmia (6.6%), sinus bradycardia (10%), tall T-wave in V2 (3.3%), pattern suggestive of acute anterior wall infarction with reciprocal changes (3.3%), myocardial ischemia (10%), non-specific ST-T changes (16.7%) and atrioventricular block (3.3%).Acute myocardial infarction complicating snake bite had been published as case reports which was not found in our series.[29,30,31,33]

ECG showing global T wave inversion in a patient with snake bite



ECG showing non specific ST-T changes in a patient with snake bite



ECHOCARDIOGRAPHY

Among 100 patients, 2 persons had hypokinesia of apical segment which recovered after a month.

RENAL FUNCTION TEST

In this series renal function test was abnormal in 10 patients of these dialysis was done in 3 patients. All patients with renal abnormalities had prolonged WBCT.[32]

SGOT AND CPK

In our series AST was elevated in 28% and CPK was elevated in 48% of patients. In study by Nayak et al[17] elevated AST titres were found in 10% of patients.

TREATMENT

All 100 patients received Anti Snake Venom, 27% were mechanically ventilated, 9% were treated with vasopressors and 3% had dialysis. Patients with pulmonary edema were treated with diuretics and mechanical ventilation.On an average patients with neurotoxic envenomation had 16 vials of ASV and patients with hemotoxic envenomation had 18 vials of ASV.

MORTALITY

Overall mortality in our study was 5%. In the five patients who died in our study, 4 patients had neurotoxic envenomation and 1 patient had neurotoxic and hemotoxic manifestations with cerebral infarction.[34] Of the 5% of death cases 2 patients had bradycardia,2 patients had features of pulmonary edema and 3 patients had hypotension along with neurotoxic and hemotoxic features.This in contrast to study by Nayak et al [17] were the mortality rate was 10% and all these patients had bleeding manifestations and abnormal electrocardiograms.

CONCLUSION

1) Snake bite is more common in 20 – 40 yrs age group in this study.

2) In our study Viperine bites were more common than Elapide bites but have a better prognosis.

3) Mortality was more among patients with neurotoxic envenomation probably due to associated autonomic instability.[20,23]

4) Cardiac involvement in snake envenomation can manifest clinically in the form of autonomic dysfunction with hypertension or hypotension, tachycardia or bradycardia, features of heart failure.[41]

5) Electrocardiographic changes in snake envenomation include cardiac rhythm disturbances and features of myocardial ischemia.

6) Prognosis was poor among patients who present with hypotension and pulmonary edema.

7) Treatment in addition to ASV requires Vasopressors and diuretics in these group of patients.

STRENGTHS AND LIMITATIONS

1) Number of patients in the study were thrice than the previous studies in cardiac manifestations in snake bite.

2) Cardiac manifestations were reported more in Elapid bites as compared to previous studies.

3) The limitations were single center study and children were not included.

4) Specific cardiac markers of myocardial injury was not done.

5) Novel echocardiographic Doppler techniques for myocardial injury was not done.

RECOMMENDATIONS

1) No specific treatment apart from snake antivenom is required for the cardiac involvement.

2) Supportive measures, such as management of pulmonary oedema with diuretics and change in ventilator strategy and treatment of dysrhythmia, may be required in specific cases.

3) Cardiac complications should be thought of when abnormal findings ensue, monitoring the patient with serial ECGs and cardiac enzymes is necessary.

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ANNEXURES

ABBREVIATIONS

ABG – Arterial blood gas analysis

AST – Aspartate Transaminase

ASV – Anti Snake Venom

BP – Blood Pressure

CPK – Creatinine Phosphokinase

FVC - Forced vital capacity

ECG - Electrocardiogram

ECHO – Echocardiography

SBC – Single Breath Count

WBCT – Whole Blood Clotting time

WHO – World Health Organisation

PROFORMA

POISON CONTROL, TRAINING AND RESEARCH CENTRE, RGGGH, CHENNAI.

Name: I.P No: DOA:

DOD:

Age : Locality: Occupation:

Gender: Male/Female Ht: cm Wt: kg BMI:

Referral:

DETAILS ABOUT SNAKE BITE:

Time Snake Bite: am/pm

Site of bite: UL/LL/R/L - Other areas specify…….

Time taken to reach the hospital:

Tourniquet tied: yes/no. If yes mention the duration…….

Nature of snake: Cobra/Krait/Russell /Saw scaled viper/others

TREATMENT BEFORE ADMISSION: HOSPITAL/NATIVE

(specify details)

CLINICAL EXAMINATION

VITALS:

HR-

BLOOD PRESSURE-supine: sitting:

RESPIRATORY RATE-

TEMPERATURE-

CVS:

RS:

ABDOMEN:

NEUROLOGICAL:

LABORATORY INVESTIGATIONS:

BIOCHEMICAL PARAMETERS

	RFT	**Day-1**	**Day -3**
1.	Sugar		
2.	Urea		
3.	Creatinine		
4.	Sodium		
5.	Potassium		

Cardiac markers

1. SGOT
2. CPK
3. CPKMB
4. LDH

LIPID PROFILE

1. Total Cholesterol
2. TGL
3. HDL

USG ABDOMEN:

DAY 1 DAY 3

ELECTROCARDIOGRAM:

ECHOCARDIOGRAPHY:

TREATMENT AND FOLLOW UP:

CONDITION ON DISCHARGE : RECOVERED/EXPIRED

If expired with informed consent of relatives,victims heart will be harvested and macroscopic examination and microscopic examination of cardiac muscle biopsy by light microscopy will be done.

S.No	Age	Sex	Duration	UL	LL	OTHER	COBRA	KRAIT	RUSSELL VIPER	SAW SCALED VIPER	DM	SHT	HEART DIS	NATIVE	HOSPITAL	TOURNIQUET	HR	BP
1	20	M	2		YES				YES							NO	92	150/90
2	25	M	2		YES			YES							YES	NO	56	90/70
3	11	M	2	YES				YES							YES	NO	130	90/68
4	27	M	2	YES			YES								YES	NO	78	110/70
5	39	M	2-4 HRS		YES		YES							YES		YES	79	130/100
6	38	M	2	YES					YES					YES		NO	60	120/80
7	16	M	2-4 HRS		YES		YES							YES		NO	90	110/70
8	30	M	2		YES		YES								YES	NO	82	110/70
9	47	F	2		YES		YES					YES			YES	NO	60	150/90
10	30	F	2-4 HRS			YES		YES								NO	56	110/76
11	70	F	2	YES			YES								YES	YES	100	110/70
12	15	M	2			YES			YES						YES	NO	100	80/60
13	47	M	2		YES		YES								YES	NO	80	130/80
14	10	F	4	YES		YES	YES									YES	108	108/70
15	75	M	2			YES		YES								NO	90	150/100
16	14	M	2		YES				YES						YES	YES	94	120/70
17	65	M	2						YES							YES	75	140/90
18	65	M	2		YES				YES							NO	78	100/70
19	25	M	4		YES				YES						YES	NO	64	130/86
20	22	M	2	YES						YES						NO	96	100/70
21	54	M	2		YES				YES						YES	YES	90	110/70
22	40	F	2		YES					YES					YES	YES	84	110/70
23	70	M	4		YES				YES						YES	YES	118	80/50
24	43	M	2		YES				YES						YES	YES	80	110/70
25	18	F	2		YES				YES					YES		YES	80	110/70
26	20	M	2		YES					YES						YES	90	110/86
27	32	M	2		YES				YES							YES	90	110/76
28	30	M	4		YES		YES									NO	58	120/70
29	40	M	2		YES				YES						YES	NO	86	110/80
30	24	M	2		YES		YES								YES	NO	72	110/70
31	39	M	2		YES										YES	NO	82	110/80
32	45	M	2	YES				YES								NO	120	90/60
33	56	F	2	YES				YES								NO	56	100/60
34	25	M	2		YES				YES							NO	86	140/90
35	46	M	4	YES						YES						YES	78	130/86
36	16	M	2	YES			YES								YES	NO	86	100/70
37	43	F	2	YES					YES		YES					NO	90	140/90
38	35	M	2	YES						YES						NO	86	120/80
39	26	F	2		YES				YES							YES	86	120/70
40	60	F	2		YES				YES							NO	78	106/80

41	25	M	2-4 HRS	YES			YES								YES	NO	82	110/78
42	62	M	2		YES							YES				NO	89	140/90
43	39	M	2			YES		YES							YES	NO	93	80/60
44	48	F	2		YES					YES	YES					NO	86	140/90
45	16	F	2	YES			YES								YES	NO	96	80/60
46	24	M	2	YES					YES							YES	86	120/80
47	36	M	2	YES					YES						YES	NO	95	108/76
48	49	M	2		YES					YES						YES	82	110/80
49	34	F	2	YES			YES								YES	NO	85	140/90
50	31	F	4	YES					YES							YES	72	120/80
51	25	M	2	YES						YES						NO	86	110/80
52	19	F	2		YES				YES							NO	86	110/80
53	23	M	2		YES				YES						YES	YES	76	110/76
54	47	M	2			YES		YES								NO	56	110/76
55	34	M	2	YES					YES							YES	79	130/86
56	29	M	2	YES			YES									NO	86	110/80
57	38	M	2		YES		YES									NO	96	130/86
58	56	F	2-4 HRS		YES										YES	NO	86	130/80
59	38	M	2	YES					YES						YES	NO	102	120/80
60	53	M	2		YES			YES							YES	NO	98	150/90
61	47	M	2		YES				YES						YES	YES	76	130/80
62	52	M	2		YES		YES								NO	NO	98	140/90
63	24	M	2-4 HRS	YES					YES						NO	YES	84	110/70
64	26	F	2		YES				YES						NO	YES	95	120/80
65	35	M	2		YES				YES						YES	YES	79	110/78
66	31	M	2	YES			YES								NO	NO	86	140/90
67	29	M	2	YES					YES						NO	NO	82	130/80
68	16	F	2		YES		YES								YES	NO	98	120/80
69	54	M	2		YES					YES		YES			NO	NO	85	140/90
70	47	M	4			YES		YES								NO	58	110/80
71	41	F	2-4 HRS		YES				YES							YES	92	110/80
72	31	M	4		YES		YES								YES	NO	80	130/80
73	38	M	4	YES					YES							NO	94	120/80
74	40	M	2	YES						YES						NO	75	140/90
75	35	M	2	YES					YES						YES	NO	86	120/76
76	45	M	4		YES			YES							YES	NO	78	110/80
77	44	M	2-4 HRS		YES				YES							NO	85	140/90
78	52	M	2		YES		YES									NO	88	130/80
79	34	F	2		YES					YES					YES	NO	98	110/86
80	27	M	2	YES					YES							YES	84	120/80
81	36	M	2			YES		YES								NO	70	110/8o
82	28	M	2	YES	YES				YES							NO	82	110/80

83	45	M	2		YES					YES					YES	YES	98	130/80
84	57	M	2	YES			YES									NO	104	140/90
85	39	M	2-4 HRS		YES				YES							YES	100	130/80
86	29	F	2		YES		YES									NO	86	110/80
87	51	F	2		YES				YES							NO	98	120/90
88	43	M	2	YES						YES						NO	102	130/80
89	55	M	2		YES				YES							NO	90	120/80
90	41	F	2-4 HRS		YES			YES								NO	58	130/80
91	27	F	4		YES				YES						YES	NO	89	130/80
92	38	M	2	YES						YES						NO	88	120/80
93	22	M	2		YES		YES								YES	NO	102	120/86
94	30	M	2		YES				YES						YES	NO	78	120/80
95	29	M	2	YES					YES						YES	NO	82	110/70
96	24	F	2		YES		YES								YES	NO	130	80/60
97	16	M	2		YES		YES								YES	YES	140	120/80
98	30	F	2		YES			YES							NO	NO	58	80/60
99	52	M	2-4 HRS	YES			YES								YES	NO	96	150/90
100	48	M	2	YES				YES							NO	NO	102	70/50

RR	NEUROTOXIC				HEMOTOXIC				LOCAL ENVENOMATION	CARDIO VASCULAR AND RESPIRATORY	
	PTOSIS	DIPLOPIA	RES PARALYSIS	NEOSTIGMINE	WBCT	BLEEDING GUMS	UGI BLEED	HEMATURIA		MURMURS	BASAL CREPTS
20	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
20	YES	YES	NO	NOT RESPONSIVE	N	NO	NO	NO	NO	NO	NO
24	YES	YES	YES	NOT RESPONSIVE	N	NO	NO	NO	NO	NO	NO
26	YES	YES	YES	RESPONSIVE	N	NO	NO	NO	YES	NO	NO
20	YES	YES	NO		N	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
18	YES	NO	NO		N	NO	NO	NO	YES	NO	NO
19	YES	NO	NO		N	NO	NO	NO	YES	NO	NO
18	YES	NO	NO		N	NO	NO	NO	YES	NO	NO
26	YES	YES	YES	NOTRESPONSIVE	N	NO	NO	NO	NO	NO	NO
20	YES	YES	NO		N	NO	NO	NO	YES	NO	NO
25	YES	YES	YES	NOTRESPONSIVE	20MINS	NO	NO	NO	YES	NO	NO
18	YES	YES	NO		N	NO	NO	NO	YES	NO	NO
19	YES	NO	NO		N	NO	NO	NO	YES	NO	NO
25	YES	YES	YES	NOTRESPONSIVE	N	NO	NO	NO	NO	NO	BASAL CREPTS
22	YES	YES	NO		20MINS	NO	NO	NO	YES	NO	NO
20	YES	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		N	NO	NO	NO	YES	NO	NO
25	YES	YES	YES		20MINS	NO	NO	YES	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	NO	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
19	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
24	YES	YES	NO	NOTRESPONSIVE	20MINS	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
20	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
19	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
18	YES	YES	NO		N	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
16	YES	YES	NO		N	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
26	YES	YES	NO		N	NO	NO	NO	NO	NO	BASAL CREPITS
25	YES	YES	YES		N	NO	NO	NO	NO	NO	NO
18	YES	NO	NO		20MINS	NO	NO	YES	YES	NO	NO
16	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
24	YES	YES	YES		N	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	NO	NO	NO
18	NO	NO	NO		20MINS	YES	NO	NO	YES	NO	NO
20	NO	NO	NO		20MINS	NO	NO	NO	NO	NO	NO

20	YES	YES	NO		N	NO	NO	NO	NO	NO	NO
20	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
24	YES	YES	YES		N	NO	NO	NO	NO	NO	BASAL CREPITS
20	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
25	YES	YES	YES		N	NO	NO	NO	YES	NO	NO
20	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
20	YES	YES	NO		20MINS	YES	NO	NO	YES	NO	NO
19	NO	NO	NO		N	NO	NO	NO	YES	NO	NO
26	YES	YES	YES		N	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	NO	NO	NO
22	YES	YES	NO		20MINS	NO	NO	NO	YES	NO	NO
20	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
24	YES	YES	YES		N	NO	NO	NO	NO	NO	NO
20	NO	NO	NO		N	NO	NO	NO	YES	NO	NO
20	YES	YES	NO		N	NO	NO	NO	NO	NO	NO
20	YES	YES	NO		N	NO	NO	NO	YES	NO	NO
20	YES	YES	NO		N	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
24	YES	YES	YES		N	NO	NO	NO	NO	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
25	YES	YES	YES		N	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	YES	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
18	YES	NO	NO		20MINS	YES	NO	YES	YES	NO	NO
20	YES	NO	NO		N	NO	NO	NO	YES	NO	NO
20	NO	NO	NO		20MINS	NO	NO	NO	NO	NO	NO
28	YES	YES	YES		N	NO	NO	NO	NO	NO	NO
17	NO	NO	NO		20MINS	NO	NO	NO	NO	NO	NO
24	YES	YES	YES		N	NO	NO	NO	NO	NO	NO
20	NO	NO	NO		20MINS	NO	NO	NO	NO	NO	NO
18	YES	YES	NO		N	NO	NO	NO	NO	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
20	NO	NO	NO		N	NO	NO	NO	YES	NO	NO
19	NO	NO	NO		20MINS	NO	NO	NO	NO	NO	NO
24	YES	YES	YES		N	NO	NO	NO	NO	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
18	YES	YES	NO		N	NO	NO	NO	YES	NO	NO
20	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	YES	NO	NO	YES	NO	NO
23	YES	YES	YES		N	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO

20	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
24	YES	YES	YES		N	NO	NO	NO	YES	NO	NO
20	YES	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
20	YES	YES	NO		N	NO	NO	NO	YES	NO	NO
20	YES	NO	NO		20MINS	YES	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		20MINS	NO	NO	NO	YES	NO	NO
27	YES	YES	YES		N	NO	NO	NO	NO	NO	NO
20	YES	YES	NO		20MINS	NO	NO	NO	YES	NO	NO
18	NO	NO	NO		N	NO	NO	NO	YES	NO	NO
24	YES	YES	NO		N	NO	NO	NO	NO	NO	NO
18	N	NO	NO		20MINS	YES	NO	NO	YES	NO	NO
18	N	NO	NO		20MINS	NO	NO	NO	NO	NO	NO
26	YES	YES	YES		N	NO	NO	NO	NO	NO	NO
25	YES	YES	YES		N	NO	NO	NO	YES	NO	NO
29	YES	YES	YES		N	NO	NO	NO	NO	NO	BASAL CREPITS
24	YES	YES	YES		N	NO	NO	NO	YES	NO	NO
25	YES	YES	YES		N	NO	NO	NO	NO	NO	NO

NO	RFT										CARDIAC MARKERS					
	SUGAR		UREA		CREATININE		SODIUM		POTASSIUM		SGOT		CPK		LDH	
	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3
1	80	94	154	40	8.5	2	134	140	4.1	3.2	145	156	250	200	708	250
2	119	96	30	32	0.8	0.8	142	140	3.9	4	24	40	120	130	200	200
3	240	110	25	25	0.7	0.7	142	132	3.6	3.5	34	36	110	120	140	120
4	145	140	36	36	1.2	1.3	135	130	4	3.8	42	46	186	183	140	146
5	90	96	32	30	0.9	0.9	140	140	3.6	3.8	154	126	126	120	130	130
6	120	136	26	28	0.9	0.9	147	140	3.8	3.6	44	46	120	114	126	125
7	120	110	18	20	0.8	0.8	134	130	3.3	3.3	129	123	120	140	120	100
8	159	178	37	47	1.1	1.3	150	146	3.3	3.3	256	200	356	250	1896	500
9	86	93	22	25	0.8	0.8	155	136	3.7	3.5	30	36	88	86	733	440
10	82	96	19	16	0.8	0.8	140	136	2.2	3.3	21	26	84	86	123	120
11	156	140	38	36	2	1.8	145	136	3.2	3.3	148	140	86	8	145	142
12	108	120	31	32	0.9	0.9	144	130	2.8	3.6	21	26	100	100	156	200
13	260	126	65	43	2.1	1.6	147	145	4.4	4	178	170	200	156	245	196
14	101	100	24	24	0.9	0.8	149	145	3.8	3.6	28	26	145	152	125	156
15	140	150	21	23	0.9	0.9	140	140	3.7	3	24	26	256	200	152	125
16	125	79	45	46	1	1	147	139	4.2	4.2	99	101	987	356	298	200
17	152	117	53	23	1.2	1	148	144	3.2	3.3	72	82	1395	588	767	608
18	172	84	56	43	1.3	1.2	140	144	3.8	4.2	110	156	1777	536	614	516
19	90	62	152	144	5.8	7.2	139	144	4.7	5.4	246	256	562	456	156	125
20	255	152	54	46	1.2	0.8	144	135	2.8	3.6	53	56	156	123	142	142
21	145	151	56	46	1.5	1.4	145	146	3.2	3.6	118	115	4260	1520	1280	456
22	258	126	40	39	0.8	0.8	144	145	3.2	3.3	256	220	145	125	156	256
23	87	56	119	98	2.3	1.3	145	148	2.7	4.1	176	145	5390	1554	1503	147
24	91	106	30	30	1.2	0.9	143	142	3.7	3.6	54	54	542	256	145	145
25	80	86	50	42	0.9	0.9	144	145	3.7	3.7	640	256	125	144	120	110
26	108	145	23	26	1	1	145	142	4.1	4	152	100	145	145	120	120
27	125	120	25	26	0.8	0.9	142	140	4.2	4	45	46	142	152	120	145
28	80	86	28	26	0.8	0.8	142	142	4	4	62	63	984	456	1272	1200
29	148	142	21	25	0.9	0.9	150	145	3.3	3.2	45	42	456	254	145	125
30	123	120	19	20	0.9	0.8	133	132	2.9	3.3	34	34	256	250	145	125
31	100	120	20	26	0.8	0.8	144	126	4.5	4	32	30	122	126	120	102
32	156	125	36	35	1.2	1	145	136	4.5	4.2	46	42	2564	1526	456	125
33	256	120	25	26	2	1.2	145	136	4.2	4	45	56	4256	1263	789	458
34	154	120	25	26	2	1	145	135	3.2	3.3	45	42	1256	1234	456	256
35	152	117	53	23	1.2	1	148	144	3.2	3.3	72	82	1395	588	767	608

NO	RFT										CARDIAC MARKERS					
	SUGAR		UREA		CREATININE		SODIUM		POTASSIUM		SGOT		CPK		LDH	
	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3
36	120	110	42	30	0.6	0.8	125	130	4.2	3.2	25	26	456	256	452	125
37	156	142	40	38	1.2	1	145	142	3.3	3	45	42	526	253	125	123
38	145	142	30	32	0.8	1	126	130	4.2	4	45	52	456	125	124	103
39	120	130	40	30	1.2	1	132	126	3.2	3	26	26	562	354	125	120
40	96	90	42	40	1.2	1	123	123	3.3	3	35	36	256	245	136	130
41	152	102	30	30	1.2	1	126	1235	4	3.3	25	25	456	254	145	112
42	145	120	36	35	1.5	1.3	123	156	3.3	3.9	24	40	236	124	111	112
43	125	111	30	36	0.8	1.2	153	126	3	3.1	24	26	4561	2561	458	246
44	236	200	40	36	1	1	142	136	3.2	3	26	25	245	200	456	245
45	123	120	40	46	1.2	2	145	146	4.2	4	45	78	452	400	457	568
46	125	120	30	30	1	1	136	135	3.6	3.3	25	24	246	240	158	168
47	156	124	76	46	2.6	1.2	145	123	5.4	4	89	102	2568	241	458	365
48	125	120	30	32	1	1.2	132	125	3	3	45	25	421	400	145	125
49	120	96	50	38	2	1	125	125	3	3.6	45	24	298	265	123	100
50	110	86	24	26	1	0.8	112	122	2.6	3.5	24	21	214	236	102	100
51	111	90	25	25	1	0.8	125	126	3	3.3	24	22	126	130	124	122
52	96	120	30	30	1.2	1	142	135	3.2	3	89	790	654	356	300	142
53	156	140	38	36	2	1.8	145	136	3.2	3.3	148	140	86	126	145	142
54	108	120	31	32	0.9	0.9	144	130	2.8	3.6	21	26	100	100	156	200
55	145	140	36	36	1.2	1.3	135	130	4	3.8	42	46	186	183	140	146
56	156	140	38	36	2	1.8	145	136	3.2	3.3	148	140	86	136	145	142
57	130	125	36	32	1	1	125	126	3.3	3	25	29	200	200	130	126
58	140	120	45	35	1	1.2	135	134	3.3	3	46	45	458	251	142	140
59	100	120	20	26	0.8	0.8	144	126	4.5	4	32	30	122	126	120	102
60	120	90	35	35	1.8	1.8	126	130	3.3	3.5	45	46	562	247	124	120
61	125	120	25	26	0.8	0.9	142	140	4.2	4	45	46	142	152	120	145
62	108	120	31	32	0.9	0.9	144	130	2.8	3.6	21	26	100	100	156	200
63	100	120	20	26	0.8	0.8	144	126	4.5	4	32	30	122	126	120	102
64	125	79	45	46	1	1	147	139	4.2	4.2	99	101	987	356	298	200
65	96	102	45	2	1.2	1.2	140	135	3.3	3	89	95	421	254	156	120
66	152	102	30	30	1.2	1	126	1235	4	3.3	25	25	456	254	145	112
67	120	96	50	38	2	1	125	125	3	3.6	45	24	298	265	123	100
68	110	101	30	32	1.6	1.2	140	125	3	3	56	53	421	254	114	124
69	145	151	56	46	1.5	1.4	145	146	3.2	3.6	118	115	4260	1520	1280	456
70	156	120	25	26	2	1.2	135	130	4.2	4	45	56	3254	1263	875	458
71	80	86	50	42	0.9	0.9	144	145	3.7	3.7	640	256	125	144	120	110
72	86	90	25	24	1	1	135	132	4	3.3	56	53	452	251	111	120

NO	RFT										CARDIAC MARKERS					
	SUGAR		UREA		CREATININE		SODIUM		POTASSIUM		SGOT		CPK		LDH	
	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3	DAY 1	DAY 3
73	145	142	30	32	0.8	1	126	130	4.2	4	45	52	456	125	124	103
74	120	136	26	28	0.9	0.9	147	140	3.8	3.6	44	46	120	114	126	125
75	100	120	20	26	0.8	0.8	144	126	4.5	4	32	30	122	126	120	102
76	256	120	25	26	2	1.2	145	136	4.2	4	45	56	4256	1263	789	458
77	145	142	30	32	0.8	1	126	130	4.2	4	45	52	456	125	124	103
78	152	102	30	30	1.2	1	126	123	4	3.3	25	25	456	254	145	112
79	102	94	154	40	1.4	1.4	134	125	4.1	3.2	145	156	250	190	708	250
80	104	93	32	25	0.8	0.8	145	136	3.7	3.5	36	36	2188	861	733	440
81	178	96	19	16	0.8	0.8	140	136	3.2	3.3	21	26	184	86	123	120
82	136	110	38	36	2	1.8	145	136	3.2	3.3	102	49	186	109	145	142
83	110	120	31	32	0.9	0.9	144	130	2.8	3.6	21	100	102	100	156	200
84	260	126	65	43	2.1	1.6	126	135	4.4	4	178	170	425	156	245	196
85	136	124	40	38	1.2	1	124	120	3.3	3	145	42	1265	253	135	123
86	98	102	38	34	2	1.8	145	136	3.2	3.3	148	140	291	142	135	142
87	145	105	58	46	1.5	1.4	146	125	3.2	3.6	118	115	4260	1520	1280	456
88	130	124	36	35	1	1	135	123	3.4	4	89	102	2568	241	458	365
89	106	90	25	28	0.8	1	125	132	3.5	3.3	56	45	241	251	111	156
90	140	110	42	30	0.6	0.8	125	130	4.2	3.2	25	26	1456	356	452	200
91	126	92	30	30	1.2	0.8	135	130	3.2	3	45	40	458	321	478	248
92	145	140	36	36	1.2	1.3	135	130	4	3.8	42	46	186	183	140	146
93	124	102	35	30	1	1	125	136	3	3.2	45	45	478	251	421	124
94	95	96	29	35	1.2	0.8	125	130	3.3	4	75	40	256	200	145	112
25	96	86	50	42	0.9	0.9	144	145	3.7	3.7	140	100	125	144	120	110
96	156	112	45	50	1.2	2	145	135	3.3	4	240	200	486	500	568	500
97	124	156	43	40	2	2	125	120	3	4	241	200	586	500	245	256
98	130	102	45	52	1.3	2	145	140	3	4	256	200	587	596	478	458
99	156	140	38	36	2	1.8	145	136	3.2	3.3	148	140	86	126	145	142
100	160	200	45	56	1.2	2	125	136	4.4	4	56	52	256	200	354	300

ELECTROCARDIOGRAM			TREATMENT GIVEN ASV						
DAY 1	DAY OF DIS	ECHO CARDIOGRAPHY	TOTAL	OUTSIDE	GH	VENTILATORY SUPPORT	VASOPRESSORS	HEMODIALYSIS	OUTCOME
WNL	WNL	NORMAL	0		21			YES	RECOVERD
BRADYCARDIA	WNL	NORMAL			35				RECOVERD
WNL	WNL	NORMAL			29	YES			RECOVERD
WNL	WNL	NORMAL			16	YES			RECOVERD
WNL	WNL	NORMAL			23	NO			RECOVERD
BRADYCARDIA	WNL	NORMAL			8				RECOVERD
T INVERSION V1-V3	WNL	NORMAL	16	8	8	NO			RECOVERD
T INVERSION V1-V3	WNL	NORMAL	26	16	10	NO			RECOVERD
BRADYCARDIA	WNL	NORMAL	16		16	NO			RECOVERD
BRADYCARDIA	WNL	NORMAL	21		21	YES			RECOVERD
WNL	WNL	NORMAL	16		16	NO			RECOVERD
WNL	WNL	NORMAL	21	8	13	YES	YES		RECOVERD
WNL	WNL	NORMAL	23	2	21	NO			RECOVERD
WNL	WNL	NORMAL	16		16	NO			RECOVERD
WNL	WNL	NORMAL	16		16	YES			RECOVERD
WNL	WNL	NORMAL	19	12	5	NO			RECOVERD
T INVERSION V1-V3	WNL	NORMAL	21		21	NO			RECOVERD
POOR PROGRESSION OF R WAVES		HYPOKINESIA LOWER 2/3 OF IVS AND LV APEX	11	11					RECOVERD
BRADYCARDIA		NORMAL	21			YES		YES	RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	21		21				RECOVERD
T INVERSION V1-V3	WNL	NORMAL	16		16				RECOVERD
RBBB	RBBB	NORMAL	21		21	YES	YES		RECOVERD
WNL	WNL	NORMAL	18		18	NO			RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
BRADYCARDIA	WNL	NORMAL	8		8				RECOVERD
DS TALL R WAVES RT PR	WNL	GR 1 DD	20	12	8	NO			RECOVERD
T INVERSION V1-V3	WNL	NORMAL	8		8	NO			RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
T INVERSION V1-V6	WNL	HYPOKINESIA LV APEX	13		13	YES	YES		RECOVERD
BRADYCARDIA	WNL	NORMAL	16		16	YES			RECOVERD
WNL	WNL	NORMAL	18		18	NO			RECOVERD
T INVERSION V1-V3	WNL	NORMAL	21		21	NO			RECOVERD

ELECTROCARDIOGRAM			TREATMENT GIVEN ASV						
DAY 1	DAY OF DIS	ECHO CARDIOGRAPHY	TOTAL	OUTSIDE	GH	VENTILATORY SUPPORT	VASOPRESSORS	HEMODIALYSIS	OUTCOME
WNL	WNL	NORMAL	16		16	YES			RECOVERD
WNL	WNL	NORMAL	13		13				RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
T INVVERSION V1-V4	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	8		8	NO			RECOVERD
WNL	WNL	NORMAL	16	8	8	NO			RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	16		16	YES	YES		RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL		16		16	YES	YES		DEATH
WNL	WNL	NORMAL	8		8				RECOVERD
T INVERSION V1-V3	WNL	NORMAL	16		16	NO		YES	RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	16		16	YES			RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	16		16	NO			RECOVERD
WNL	WNL	NORMAL	16		16	NO			RECOVERD
BRADYCARDIA	WNL	NORMAL	16	8	8	YES			RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	16		16	NO			RECOVERD
T INVERSION V1-V3	WNL	NORMAL	16		16	NO			RECOVERD
PR PROLONGATION	WNL	NORMAL	16		16	NO			RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	16	8	8	YES			RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	16	8	8	YES			RECOVERD
WNL	WNL	NORMAL	15	8	5				RECOVERD
WNL	WNL	NORMAL	8		8	NO			RECOVERD
WNL	WNL	NORMAL	21		21	NO			RECOVERD
WNL	WNL	NORMAL	8	0	8	NO			RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	16		16	YES			RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
BRADYCARDIA	WNL	NORMAL	16		16	YES			RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	16	8	8	NO			RECOVERD

ELECTROCARDIOGRAM			TREATMENT GIVEN ASV						
DAY 1	DAY OF DIS	ECHO CARDIOGRAPHY	TOTAL	OUTSIDE	GH	VENTILATORY SUPPORT	VASOPRESSORS	HEMODIALYSIS	OUTCOME
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL			8				RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	16		16	YES			RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	16	8	8	NO			RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
POOR PROGRESSION OF R WAVES RT PREC LEADS	WNL	NORMAL	21		21	NO			RECOVERD
WNL	WNL	NORMAL	16		16	YES			RECOVERD
WNL	WNL	NORMAL	8		8	NO			RECOVERD
WNL	WNL	NORMAL	8	0	8				RECOVERD
WNL	WNL	NORMAL	16	0	16	YES			RECOVERD
WNL	WNL	NORMAL	16		16				RECOVERD
WNL	WNL	NORMAL	16		16	NO			RECOVERD
WNL	WNL	NORMAL	21		21				RECOVERD
WNL	WNL	NORMAL	8		8	NO			RECOVERD
WNL	WNL	NORMAL	16	8	8	NO			RECOVERD
BRADYCARDIA	WNL	NORMAL	16		16	YES			RECOVERD
POOR PROGRESSION OF R WAVES	WNL	NORMAL	13	5	8	NO			RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL	NORMAL	16		16	NO			RECOVERD
WNL	WNL	NORMAL	13	8	5	NO			RECOVERD
WNL	WNL	NORMAL	8		8				RECOVERD
WNL	WNL		16		16	YES	YES		DEATH
WNL	WNL		16	8	8	YES	YES		DEATH
BRADYCARDIA	WNL		16		16	YES	YES		DEATH
WNL	WNL	NORMAL	16		16	YES			RECOVERD
WNL	WNL		16		16	YES	YES		DEATH

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI -3

Telephone No: 04425305301
Fax : 044 25363970

CERTIFICATE OF APPROVAL

To
Dr. Vinoth Kumar.S
PG in MD General Medicine
Madras Medical College, Chennai -3.

Dear Dr. Vinoth Kumar.S

The Institutional Ethics Committee of Madras Medical College reviewed and discussed your application for approval of the proposal entitled " Cardiac involvement in snake envenomation " No. 01042011.

The following members of Ethics Committee were present in the meeting held on 21.04.2011 conducted at Madras Medical College, Chennai -3.

1.	Prof. S.K. Rajan, MD	-- Chairperson
2.	Prof. V. Kanagasabai MD Dean, Madras Medical College, Chennai-3,	-- Deputy chairman
3.	Prof. A. Sundaram, MD Vice Principal , Madras Medical College, Chennai -3	-- Member Secretary
4.	Prof R. Sathianathan MD	-- Member
5.	Prof R. Nandhini, MD Director, Institute of Pharmacology, MMC, Ch-3	-- Member
6.	Prof. Pregna B. Dolia MD Director , Institute of Biochemistry, MMC, Ch-3	-- Member
7.	Prof. C. Rajendiran .MD Director , Institute of Internal Medicine, MMC, Ch-3	-- Member
8.	Thiru. A. Ulaganathan Administrative Officer, MMC, Chennai -3	-- Layperson
9.	Thiru. S. Govindasamy . BA.BL	-- Lawyer
10.	Tmt. Arnold Soulina	-- Social Scientist

We approve the proposal to be conducted in its presented form.

Sd /. Chairman & Other Members

The Institutional Ethics Committee expects to be informed about the progress of the study, any SAE occurring in the course of the study, any changes in the protocol and patient information / informed consent and asks to be provided a copy of the final report

Member Secretary, Ethics Committee