CLINICAL PROFILE OF SCORPION ENVENOMATION WITH ELECTROCARDIOGRAPHIC CHANGES AND CARDIAC BIOMARKERS

DISSERTATION SUBMITTED

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DEPARTMENT OF GENERAL MEDICINE

TIRUNELVELI MEDICAL COLLEGE HOSPITAL

TIRUNELVELI – 627011

MAY-2023

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"CLINICAL PROFILE OF SCORPION ENVENOMATION WITH ELECTROCARDIOGRAPHIC CHANGES AND CARDIAC BIOMARKERS"** is a bonafide original work done by **Dr. ANANTHARAJ A,** Post Graduate in the Department of General Medicine, Tirunelveli Medical College and Hospital, Tirunelveli, in partial fulfilment of the requirement for the award of M.D. Degree in General Medicine (Branch I) under the Tamilnadu Dr. M.G.R Medical University, Chennai.

For 16.12.22

Prof. Dr. S ALAGESAN. MD DM,

Professor & Head of the Department,

Department of General Medicine,

Tirunelveli Medical College.

Dr. S. Alagesan, MD.,
Reg. No. 43658
Prof. & HOD of Medicine
Tirunelveli Medical College Hospital,
Tirunelveli.

Prof. Dr. A RAJESH MD

Unit chief,

Department of General Medicine,

Tirunelveli Medical College.

Dr. A. Rajesh MD.,
Reg No. 56270
Professor of Medicine
Tirunelveli Medical College
Tirunelveli

CERTIFICATE BY THE GUIDE

This is to certify that this dissertation **"CLINICAL PROFILE OF SCORPION ENVENOMATION WITH ELECTROCARDIOGRAPHIC CHANGES AND CARDIAC BIOMARKERS"** is a bonafide original work done by **Dr. ANANTHARAJ A,** Post Graduate student in the department of General Medicine, Tirunelveli Medical College and Hospital, Tirunelveli, under my guidance during the academic period of 2020-2023.

Prof. Dr. A RAJESH MD,

Department of General Medicine

Tirunelveli Medical College,

Tirunelveli.

Dr. A. Rajesh MD.,
Reg No. 56270
Professor of Medicine
Tirunelveli Medical College
Tirunelveli

CERTIFICATE

This is to certify that the Dissertation entitled **"CLINICAL PROFILE OF SCORPION ENVENOMATION WITH ELECTROCARDIOGRAPHIC CHANGES AND CARDIAC BIOMARKERS"** presented herein by **Dr. ANANTHARAJ A,** is an original work done in the Department of General Medicine, Tirunelveli Medical College and Hospital, Tirunelveli for the award of the Degree of M.D. General Medicine (Branch I) under my guidance and supervision during the academic period of 2020 -2023.

Dr. M. RAVICHANDRAN M.D,

The DEAN,

Tirunelveli Medical College,

Tirunelveli - 627011.

DEAN
TIRUNELVELI MEDICAL COLLEGE
TIRUNELVELI - 11

DECLARATION

I solemnly declare that the dissertation entitled **"CLINICAL PROFILE OF SCORPION ENVENOMATION WITH ELECTROCARDIOGRAPHIC CHANGES AND CARDIAC BIOMARKERS"** is an original work done by me at the department of General Medicine, Tirunelveli Medical College and Hospital, Tirunelveli, under the guidance and supervision of **Prof. Dr. A RAJESH MD,** Department of General Medicine, Tirunelveli Medical College and Hospital, Tirunelveli. The dissertation is submitted to The Tamilnadu Dr.M.G.R. Medical University, Chennai towards the partial fulfilment of the requirements for the award of M.D. Degree in General Medicine (Branch I).

Dr ANANTHARAJ A
Registration No : 200120104003
Post Graduate Student,
M.D General Medicine,
Department of General Medicine,
Tirunelveli Medical College.

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I am also grateful to all the patients who were willing to participate in this study without whom this would not have been possible.

I dedicate this work to my family who have been a source of constant support and inspiration to me. And finally, I thank God the almighty for the blessings and opportunities given to me.

TIRUNELVELI MEDICAL COLLEGE

INSTITUTIONAL RESEARCH ETHICS COMMITTEE

TIRUNELVELI, STATE OF TAMILNADU, SOUTH INDIA PIN 627011
91-462 2572731-EXT; 91-462 2572844; 91-462-2579785 91-462 2572811 16
[illegible]

CERTIFICATE OF REGISTRATION & APPROVAL OF THE TIREC REF NO :1999/GM/2021

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PRINCIPAL INVESTIGATOR: DR. ANANTHA RAJ MBBS
DESIGNATION OF PRINCIPAL INVESTIGATOR: RESIDENT
DEPARTMENT & INSTITUTION: GENERAL MEDICINE, TIRUNELVELI MEDICAL COLLEGE, TIRUNELVELI

Dear DR. ANANTHA RAJ MBBS, Tirunelveli Medical College Institutional Ethics Committee (TIREC) reviewed and discussed your application during The IEC meeting held on 06.07.2021.

THE FOLLOWING DOCUMENTS WERE REVIEWED AND APPROVED

1.	TIREC Application Form	☐
2.	Study Protocol	☐
3.	Department Research Committee Approval	☐
4.	Patient Information Document and Consent Form in English and Vernacular Language	☐
5.	Investigator's Brochure	☐
6.	Proposed Methods for Patient Accrual Proposed	☐
7.	Curriculum Vitae of The Principal Investigator	☐
8.	Insurance /Compensation Policy	☐
9.	Investigator's Agreement with Sponsor	☐
10.	Investigator's Undertaking	☐
11.	DCGI/DGFT approval	☐
12.	Clinical Trial Agreement (CTA)	☐
13.	Memorandum of Understanding (MOU)/Material Transfer Agreement (MTA)	☐
14.	Clinical Trials Registry-India (CTRI) Registration	☐

THE PROTOCOL IS APPROVED IN ITS PRESENTED FORM ON THE FOLLOWING CONDITIONS

1. The approval is valid for a period of 2 year/s or duration of project whichever is later
2. The date of commencement of study should be informed
3. A written request should be submitted 3weeks before for renewal / extension of the validity
4. An annual status report should be submitted.
5. The TIREC will monitor the study
6. At The time of PI's retirement/leaving the institute, The study responsibility should be transferred to a person cleared by HOD
7. The PI should report to TIREC within 7 days of the occurrence of the SAE. If the SAE is Death, the Bioethics Cell should receive the SAE reporting form within 24 hours of the occurrence.
8. In the events of any protocol amendments, TIREC must be informed and the amendments should be highlighted in clear terms as follows:
   a. The exact alteration/amendment should be specified and indicated where the amendment occurred in the original project. (Page no. Clause no. etc.)
   b. The PI must comment how proposed amendment will affect the ongoing trial. Alteration in the budgetary status, staff requirement should be clearly indicated and the revised budget form should be submitted.
   c. If the amendments require a change in the consent form, the copy of revised Consent Form should be submitted to Ethics Committee for approval. If the amendment demands a re-look at the toxicity or side effects to patients, the same should be documented.
   d. If there are any amendments in the trial design, these must be incorporated in the protocol, and other study documents. These revised documents should be submitted for approval of the IEC, only then can they be implemented.
   e. Approval for amendment changes must be obtained prior to implementation of changes.
   f. The amendment is unlikely to be approved by the IEC unless all the above information is provided.
   g. Any deviation/violation/waiver in the protocol must be informed.

STANDS APPROVED UNDER SEAL

Dr.K.Shantaraman, MD
Member Secretary, TIREC
Tirunelveli Medical College, Tirunelveli – 627011
State of Tamilnadu, South India

CERTIFICATE – II

This is to certify that this dissertation work titled **"CLINICAL PROFILE OF SCORPION ENVENOMATION WITH ELECTROCARDIOGRAPHIC CHANGES AND CARDIAC BIOMARKERS"** of the candidate **Dr. ANANTHARAJ A** with registration number **200120104003** for the award of **M.D. Degree** in the branch of **GENERAL MEDICINE (BRANCH I).** I personally verified the urkund.com website for the purpose of plagiarism check. I found that the uploaded thesis file contains from introduction to conclusion page and result shows **0 percentage** of plagiarism in the dissertation.

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Dr. A. Rajesh MD.,
Reg.No. 56270
Professor of Medicine
Tirunelveli Medical College
Tirunelveli

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Similarity	0%
Analysis address	rajanantha94.tnmg@analysis.urkund.com

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INTRODUCTION Scorpion (Mesobuthus tamulus) envenomation is a frequently encountered medical condition in our area. Most bites are harmless, eventhough some may produce significant morbidity and occasional mortality(1). Cardiovascular manifestations are more common following indian red scorpion envenomation and myocarditis is the most lethal complication, and is the major cause of death. As the criteria for myocyte injury in scorpion sting is not well established, the measurement of cardiac biomarkers and ECG monitoring may augment the clinical diagnosis in scorpion envenomation.(2)

AIMS AND OBJECTIVES OF THE STUDY To study the clinical profile and Cardiovascular manifestations following scorpion sting • To study the electrocardiographic changes seen following scorpion sting • To assess the significance of serum levels of CPK-MB and SGOT as biomarkers of myocardial injury • To study the correlation between these biomarkers and ECG changes and clinical status following scorpion envenomation

REVIEW OF LITERATURE Hottentotta tamulus

Fig 1.1,. Indian red scorpion (Mesobuthus tamulus) Kingdom: Animalia Phylum: Arthropoda Subphylum: Chelicerata Class: Arachnida Order: Scorpiones Family: Buthidae Genus: Hottentotta Species: H. tamulus Scorpion envenomation is a threat to more than 2 billion people worldwide with annual sting number exceeding one million. As observed in other parts of the world, scorpion stings are serious public health problem not only because of their high incidence, but also because of their potential to cause severe (sometimes fatal) signs and symptoms, especially in children world wide.(3)(4)

Approximately 1,500 scorpion species belonging to 18 families had been described now. However, only 30 species are considered dangerous to man, with 29 of them belonging to the family Buthidae. 11 account for serious and fatal envenoming, including scorpions of the genus Androctonus and Buthus in North Africa, Leiurus in the Middle East, Tityus in South America, Centruroides in North and Central America, Mesobuthus in Asia (especially in India), and Parabuthus in South Africa. (5) In Brazil, medically important scorpions are belong to Tityus genus; the major envenoming-related species are T. serrulatus and T. bahiensis in the Southeast, T. stigmurus in the Northeast, and T. obscurus (paraensis) in the North Most stings involve mild envenoming, while more serious envenoming caused by T. serrulatus(6)

List Of Few Most Dangerous and Unique Scorpions 1. Indian Red Scorpion (Hottentotta Tamulus) 2. Deathstalker Scorpion (Leiurus Quinquestriatus) 3. Arabian Fat-Tailed Scorpion (Androctonus Crassicauda) 4. Yellow Fat-Tailed Scorpion 5. Black Spitting Thick-Tailed Scorpion 6. Striped Bark Scorpions

Fig,. 1.2 Deathstalker Scorpion (Leiurus Quinquestriatus)

Fig,. 1.3 Arabian Fat-Tailed Scorpion (Androctonus Crassicauda)

CONTENT

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REVIEW OF LITERATURE

Hottentotta tamulus	
	
Fig 1.1,. Indian red scorpion (Mesobuthus tamulus)	
Kingdom:	**Animalia**
Phylum:	**Arthropoda**
Subphylum:	**Chelicerata**
Class:	**Arachnida**
Order:	**Scorpiones**
Family:	**Buthidae**
Genus:	**Hottentotta**

Species:	**H. tamulus**

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List Of Few Most Dangerous and Unique Scorpions

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Fig,. 1.2 Deathstalker Scorpion (Leiurus Quinquestriatus)

Fig,. 1.3 Arabian Fat-Tailed Scorpion (Androctonus Crassicauda)

Fig 1.5,. Black Spitting Thick-Tailed Scorpion

The purpose of this review to characterize the scorpion-related cardiomyopathy, and to clarify its pathophysiological mechanisms,

and to describe potentially useful treatments in this particular context. (12)

Early cardiovascular dysfunction

- vascular phase of scorpion envenomation-catecholamine-related vasoconstriction leading to a sharp increase in left ventricular (LV) afterload, thereby impeding LV emptying, and increasing LV filling pressure. (13)
- myocardial phase occurs, characterized by striking alteration in LV contractility (myocardial stunning), low cardiac output, and hypotensive state. The right ventricle involvement is symmetric to that of LV with a profound and reversible alteration in right ventricular performance. This phase is unique that it is reversible spontaneously or under inotropic treatment.(14)
- Scorpion myocardiopathy combines the features of takotsubo myocardiopathy (or stress myocardiopathy) which is linked to a massive release in catecholamines leading to myocardial ischemia through coronary vasomotor abnormalities (epicardial coronary spasm and/or increase in coronary microvascular resistance),

- Acute heart failure presenting as cardiogenic shock or pulmonary edema, or both is the most severe presentation of scorpion envenomation accounting for 0.27% lethality rate.(14)
- Treatment of pulmonary edema due to scorpion envenomation follows the same principles as those applied for the treatment of cardiogenic pulmonary edema in general: this begins with oxygen supplementation targeting an oxygen saturation of 92% or more, by oxygen mask, continuous positive airway pressure, noninvasive ventilation, or conventional mechanical ventilation. Dobutamine effectively improves hemodynamic parameters and may reduce mortality in severe scorpion envenomation(17)(18).

Characterization of Venom and Analyses of Sequence-Structure-Functional Impacts

Biochemical and Proteomic Characterization

Scorpion venomis a cocktail of enzymatic and non-enzymatic proteins

Enzymatic protein

i) short toxins are comprised of 30–40 amino acids

ii) long toxins have 60–70 amino acids ,

Non-enzymatic toxins can be divided into four groups based on their biological functions and pharmacological activity, namely

i. Sodium channel toxin
ii. Chloride channel toxin
iii. Potassium channel toxin
iv. Calcium channel toxin

The last decade has seen expansion of research techniques utilized to identify, characterize, and quantify the venom composition of venomous animals. Traditional approaches have relied on biochemical analyses of venome enzymes and venome profiling by SDS-PAGE and gel filtration chromatography. However, recently, these techniques have been coupled with high-throughput genomic, transcriptomic, and proteomics approaches to give a more profound and comprehensive analyses of a species. Several studies drawn a good correlation between venom composition with toxicity and pathophysiology of sting.(20)

liquid chromatography-mass spectrometry (LC-MS/MS)-based proteomics combined with biochemical and *in vitro* pharmacological activity assay to characterize the venom composition of the Indian red scorpion. Proteomic identified 110 proteins and polypeptides belonging to 13 protein families. The venom had a predominant of ion channel toxins (Na^+ and K^+ channels affecting toxins). Other minor venom components are serine protease-like protein, serine protease inhibitor, antimicrobial peptide, hyaluronidase, makatoxin, lypolysis potentiating peptides, neurotoxin affecting Cl^- channels, parabutoporin, Ca^{2+} channel toxins, bradykinin potentiating peptides, HMG CoA reductase inhibitor, and other toxins with unknown pharmacological activity. Further, the low molecular weight insect-selective toxins BtTx3 (3,796 Da) and ButaIT (3,856.7 Da) was identified. These toxins can be developed as insecticidal agents against lepidopteran insect species (21).

Indian red scorpion venom was devoid of *in vitro* hemolytic activity, and also failed to interfere with blood coagulation and platelet modulation (activation or deaggregation) under *in vitro* conditions.The 3D structure of some of the toxins deposited in UniProt . The occurrence of several

other venom toxins from different Mesobuthus and Heterometrus species also found throughout the Indian subcontinent.

The scorpion venom is water soluble complex mixture of neurotoxin, cardiotoxin, nephrotoxin, hemolysins, phosphodiesterases, phospholipase, hyaluronidases, histamine and chemicals. The venom can cause myocardial damage by several pathogenetic mechanisms.(25)(27)(28)

Myocardial ischemia by coronary spasm:

Release of vasoactive, inflammatory and thrombogenic peptides and amine constituents (histamine, serotonin, bradykinin, leukotrienes, thromboxane), which act on the coronary vasculature and induce coronary artery vasospasm and facilitate platelet aggregation as well as thrombosis.(26)

Direct cardiotoxic effect of the venom causing toxic myocarditis by reduction of Na-K-ATPase and adrenergic myocarditis by releasing adrenaline and noradrenaline from neurons, ganglia and adrenals, thereby

increasing myocardial oxygen demand direct inotropic and chronotropic effect on already compromised myocardial blood supply.(30)

Anaphylactic reaction:

Release of allergenic proteins causes anaphylactic shock that leading to hypotension with vasodilation and decreased of intravascular volume with reduced myocardial perfusion. Scorpion venom inhibits angiotensin converting enzyme (ACE), which results in accumulation of bradykinin, and is implicated in the development of pulmonary edema. Bahloul et al examined the histopathology of two fatal myocarditis causes resulting from a scorpion bite, revealed a mixed picture of toxic myocarditis and coagulative myocytolysis, similar to catecholamine-induced cardiomyopathy.(32)

TABLE 1. **Comparative list of the pharmacological effects induced by toxins from Indian red scorpion venoms of different geographical region.**

Sl. No	Pharmacological effects	Responsible toxins	Geographical region				References
			Southern India	Western India	Northern India	Sri Lanka	
1	Vomiting, nausea	Na^+ channel toxin (α neurotoxin)	YES (Yuvaraja et al., 2019)	YES (Bawaskar and Bawaskar, 1998)	YES (Agrawal et al., 2015)	Not known	Jimenez et al. (2008)
2	Sweating	Na^+ channel toxin (α neurotoxin)	YES (Yuvaraja et al., 2019)	YES (Bawaskar and Bawaskar, 1998)	YES (Agrawal et al., 2015)	YES (Kularatne et al., 2015)	Jimenez et al. (2008)
3	Salivation	Na^+ channel toxin (α neurotoxin)	Not known	YES (Bawaskar and Bawaskar, 1998)	Not known	YES (Kularatne et al., 2015)	Jimenez et al. (2008)
4	Bradycardia, hyperkalamia, vasoconstriction	Na^+ channel toxin (α neurotoxin)	Not known	Not known	Not known	Not known	Jimenez et al. (2008); Tiwari and Deshpande, (1996)
5	Tachycardia	Na^+ channel toxin (α neurotoxin)	YES (Bawaskar and Bawaskar, 1998; Madhavan, 2015; Yuvaraja et al., 2019)	YES (Bawaskar and Bawaskar, 1996)	Not known	YES (Kularatne et al., 2015)	Jimenez et al. (2008)
6	Pulmonary oedema	Na^+ channel toxin (α neurotoxin)	YES (Bawaskar and Bawaskar, 1998; Madhavan, 2015; Yuvaraja et al., 2019)	YES (Bawaskar and Bawaskar, 1996)	Not known	Not known	Jimenez et al. (2008)
7	Chest pain	Na^+ channel toxin (β neurotoxin)	YES (Yuvaraja et al., 2019)	Not known	YES (Agrawal et al., 2015)	Not known	Stevens et al. (2011)
8	Breathlessness, cough	Na^+ channel toxin (β neurotoxin)	Not known	Not known	YES (Agrawal et al., 2015)	Not known	Stevens et al. (2011)
9	Cardiac arrythmias	K^+ channel toxin	Not known	YES (Bawaskar and Bawaskar, 1998)	Not known	Not known	Ravens and Cerbai, (2008)
10	Hypotension	Bradykinin potentiating peptide	YES	Not known	Not known	YES (Kularatne et al., 2015)	Ianzer et al. (2004)
11	Hypertension	Ca^{2+} channel toxin	YES (Yuvaraja et al., 2019)	YES (Bawaskar and Bawaskar, 1996)	Not known	YES (Kularatne et al., 2015)	Fan et al. (2015); Touyz et al. (2018)
12	Priapism	Bukatoxin and Makatoxin	YES (Madhavan, 2015; Yuvaraja et al., 2019)	YES (Bawaskar and Bawaskar, 1998)	Not known	Not known	Gibson and McFadzean (2001)
13	Piloerection	Not characterized	YES (Yuvaraja et al., 2019)	Not known	Not known	YES (Kularatne et al., 2015)	–
14	Myocarditis	Na^+ channel toxin (α neurotoxin)	YES (Bawaskar and Bawaskar, 1998; Madhavan, 2015; Yuvaraja et al., 2019)	Not known	Not known	Not known	Jimenez et al. (2008)

PHYSIOPATHOLOGY OF ENVENOMING

The venom of Buthidae family scorpions has several low-molecular weight proteins (neurotoxins) that acting mainly on two classes of ion channels: the sodium (Na+) and potassium (K+) voltage-gated channels. These channels will conduct the electrical impulse in most excitable tissues, promoting permeability to ions, which initiates the action potential. Alpha and beta toxins act on Na+ channels at two pharmacologically distinct sites: alpha toxins bind to receptor site-4 and inhibit channel inactivation while the beta toxins bind receptor-type 3 and enhance activation of the channel upon subsequent depolarization. Toxins acting on K+ channels physically block them, and prevent ionic conduction, thus prolonging the action potential. Therefore, the Na+ and K+ channel toxins synergize to cause intense and prolonged depolarization, leading to neuronal excitation. This in turn stimulates postganglionary nerve endings of the sympathetic and parasympathetic nervous system and of the adrenal medulla, inducing the release of acetylcholine, adrenaline, and noradrenaline. These mediators act rapidly after the sting to initiate a chain of events that represents scorpion

envenoming, triggering the onset of clinical manifestations in practically all systems of the organism. Recent animal and human studies have identified other mediators in the physiopathology of scorpion envenoming, such as interleukins, tumor necrosis factor (TNF), platelet activating factor (PAF), and activation of the complement system and of substances such as endothelin-1 and neuropeptide Y, which may contribute to seriousness of the case(38)(39)(40)

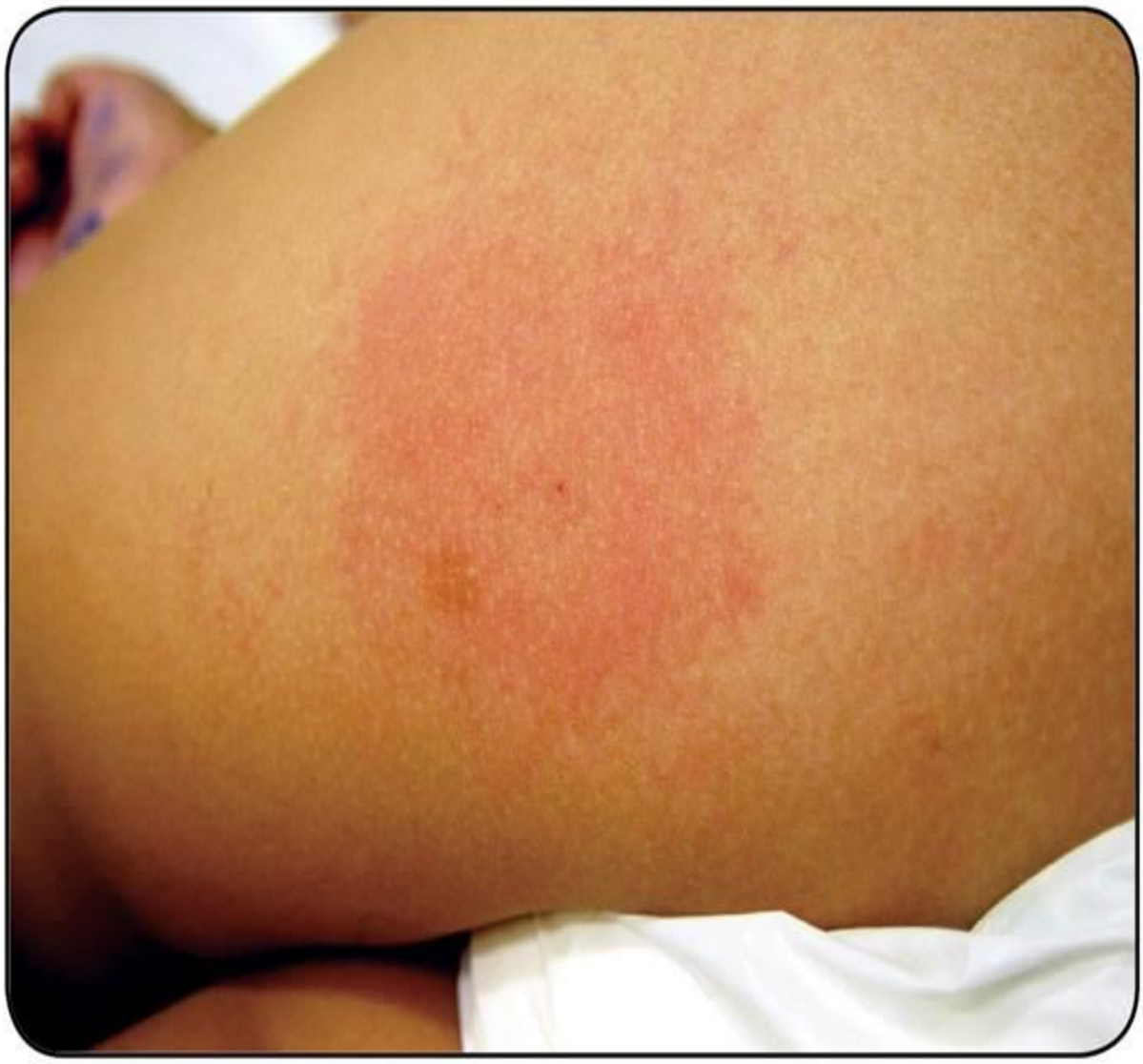
CLINICAL SIGNS AND SYMPTOMS

Local

Erythema and discrete edema are observed at the sting site, and the point of inoculation is usually difficult to locate.

Piloerection, diaphoresis, and chills be localized to the site or to the entire limb is involved. Local pain is practically immediate of varying intensity ranging from mild to very intense or unbearable, depending mainly on individual sensitivity and irradiating to the root of the limbs. It is characterized by tingling, burning, or stinging. Regardless of severity

of envenoming, pain and paresthesia may persist at the site or at the affected limb for several days.(43)



FIG,. 2.0 - Erythema and inoculation point of a Tityus serrulatus serrulatus sting in the thigh.

Systemic manifestations:

The initial clinical manifestations are mainly because of venom induced cholinergic and adrenergic effects.Acetylcholine release causes myosis, bradycardia, cardiac arrhythmias, arterial hypotension, increased lachrymal, nasal, salivary, pancreatic, gastric and bronchial secretions, diaphoresis, tremors, piloerection and muscle spasms, and thus increases blood amylase levels. Manifestation secondary to catecholamine release include mydriasis, cardiac arrhythmias, tachycardia, arterial hypertension, acute pulmonary edema (APE), cardiac failure, and shock. Adrenergic firing causes hyperglycemia and leukocytosis and contributes to hypopotassemia.

Patient signs and symptoms are variable; the established clinical picture depends on quantity of mediators released, and on the relative contribution of acetylcholine or adrenaline, which often antagonistic. Symptoms usually starts with more transitory parasympathetic activation. Severity is generally determined by the long-lasting effects of high catecholamine concentrations in cardiovascular system

Majority of the species of scorpion from Buthidae family causes similar reactions, except for Tityus obscurus in Brazil, Centruroides, and Parabuthus, which induce neurological manifestations. Signs and symptoms of envenoming caused by T. obscurus in some regions of Pará (Santarém) causes myoclonus (a sensation of an electric shock throughout the body), dysmetria, dysarthria, ataxia, paresthesias and hyperrefl exia, compatible with acute cerebellar syndrome with abnormal muscle movements, in addition to rhabdomyolysis and acute renal failure.(22)

CLASSIFICATION OF SERIOUSNESS

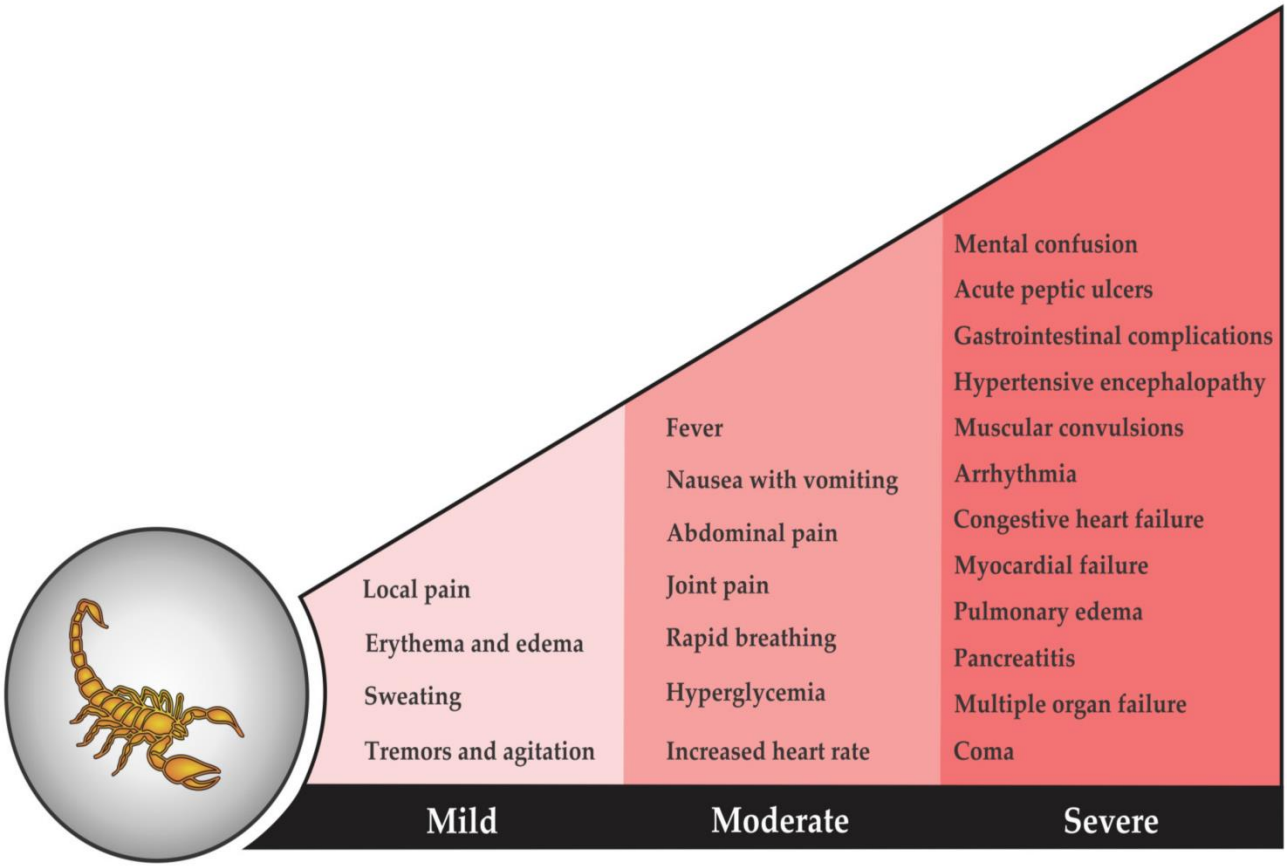


Fig 2.1

For therapeutic and prognostic guidance, envenoming is classified as mild, moderate and serious depending on intensity of the initial symptoms.

Mild

Presence of local manifestations: mild nausea, agitation, and tachycardia may be present, and are related to pain. These represent the great majority of envenoming episodes.

Moderate

In addition to local symptoms, some of the low intensity systemic manifestations may occur: diaphoresis, nauseas, some vomiting episodes, tachycardia, tachypnea, agitation, and arterial hypertension.

Serious

Systemic manifestations are evident and intense: numerous vomiting episodes, excessive salivation, profuse diaphoresis, hypothermia, tachydyspnea, bronchorrhea, tachy or bradyarrythmias, arterial hyper- or hypotension, alternating agitation and prostration, and, rarely, muscle spasms and convulsions. Progression to cardiac failure, APE, shock and death may occur. The local pain is usually masked by the above signs and symptoms, and may later reappear with the improvement of signs and symptoms. Convulsions related to hypoxia or to arterial hypertension

and ischemic cerebrovascular accidents have also been described and are quite rare. The severity of envenoming usually manifests within the first two hours after the sting, i.e., a patient is in a serious condition since the beginning, with the early occurrence of numerous vomiting episodes (a premonitory sign of seriousness) and with progression to systemic manifestations. In the most serious cases, when intense catecholamine release occurs, there is early cardiac aggression.(26)

COMPLICATIONS OF SCORPION ENVENOMING

Envenoming severity is related to cardiac and hemodynamic changes triggered along with cardiogenic shock and APE being the major cause of death Physiopathology of cardiac involvement and the etiology of the APE in scorpion haves been debated since 1980s

Cardiac involvement, usually is reversible within the first week post-sting, has been mainly attributed to the adrenergic firing induced by the scorpion toxin. Catecholamine-mediated cardiac damage seems to multifactorial and it attributed to relative hypoxia caused by the increase in heart rate, or by coronary spasm and vasoconstriction of the microcirculation, in addition to direct toxic effect of the mediators on

myocardial cells through the increase in intracellular calcium. Increased levels of pro inflammatory cytokines and the neuropeptides endothelin-1 and neuropeptide Y (these in experimental animals) detected in scorpion envenoming may also potentiate myocardial dysfunction through their direct depressive effect in contractile cardiac function and by causing coronary constriction, respectively, There is an experimental evidence of a direct toxic effect of the venom on inotropic properties of the heart, although this alone don't explain the reduced systolic performance. The cardiac involvement observed manifests clinically mainly a reversible acute left ventricle (LV) dysfunction of varying degrees of severity, with altered global or regional mobility occurring soon after the sting, and possible progression to APE. It is accompanied by increased serum levels of cardiac markers, and also changes in the electrocardiogram (ECG) and echocardiogram (ECHO) readouts. Another characteristic of the cardiac dysfunction mediated by catecholamine concerns the ECHO, whose findings dont follow the anatomic distribution of the coronary arteries. The first description of myocardial perfusion after scorpion envenoming was published by Gueron et al. who reported regional perfusion defects

in resting myocardial scintigraphy (201Thallium) in 14-year-old patients with serious scorpion envenoming, APE, and a 33% LV ejection fraction (LVEF), 2h after the sting. These changes regressed after 72h, later described perfusion changes compatible with LV dysfunction in 6 patients (3 children and 3 adults) 12-74h after Androctonus australis envenoming; these changes also correlated with ECHO findings. Exams repeated after 6 and 15 days in two patients showed partial regression of these findings. In Brazil, changes in myocardial perfusion scintigraphy (99Technetium) in 12 patients aged 1 to 12 years with serious T. serrulatus envenoming were observed during the first 72h after the sting. The topography and intensity of these changes also correlated with those detected by ECHO, and were not consistent with the territory of coronary artery distribution(44) (45). The intensity of cardiac dysfunction was correlated with the severity of envenoming, since 6 of the 7 patients with LVEF < 35% (severe dysfunction) had APE, as opposed to only 1 patient with LVEF ≥ 35%, corroborating the cardiogenic etiology of APE. Control exams performed on all patients were practically normal. These reported changes of perfusion are compatible with the occurrence of

spasm of the coronary microcirculation, supporting the role of adrenergic hyperstimulation. Several other clinical situations in addition to scorpion envenoming are associated with excessive concentrations of catecholamines released through an endogenous pathway, as is the case for pheochromocytoma, subarachnoid hemorrhage, and stress-induced cardiomyopathy, or by an exogenous pathway as is the case for accidental administration of high epinephrine doses. In all of these conditions, the cardiopulmonary manifestations are similar to those observed in scorpion envenoming. We recently reported cardiac magnetic resonance (CMR) findings for a 7-year-old patient with serious T. serrulatus envenoming associated with APE. Apical LV ballooning was observed in association with 29% LVEF; this was accompanied by global edema of the middle and apical myocardium. Full recovery of apical region mobility and of LVEF (60%), just like of the global edema occurred and was observed in CMR repeated 7 months later. These findings are similar to those observed in takotsubo cardiomyopathy, suggesting that excess catecholamine release may be the common mechanism underlying the physiopathology of cardiac dysfunction in

these two situations. Regarding APE, available evidence strongly indicates a cardiogenic etiology, as described above, although it cannot be ruled out that non-cardiogenic factors may also contribute to this complication. In general, APE develops within the first hours after envenoming, possibly occurring during the first 24h.(26)(27)

Investigation

COMPLEMENTARY EXAMS :

Complementary exams are performed only in patients with moderate and serious envenomings, and in most cases are reversible in the first week, depending on the severity. The biochemical exams normalize as during the first hours after scorpion antivenom (SAV).

Blood :

Hyperglycemia, leukocytosis, and hypopotassemia occur early, and an increase in blood amylase level is observed in a large percentage of cases.

When cardiac involvement occurs, there is an increase in the enzymes phosphocreatine kinase CK-MB, glutamic oxaloacetic

transaminase (GOT), lactic dehydrogenase (LDH), troponin I, and aminoterminal pro-brain natriuretic peptide (NT-proBNP) in serial determination, similar to the profile detected in myocardial infarction. A disorder of acid-base equilibrium of the mixed type is usually observed; initially, this presents as metabolic acidosis and respiratory alkalosis, and may progress to respiratory acidosis.

The presence of hyperglycemia is a useful early finding when there is no history or certainty about the episode of envenoming (in the case of children, they may commonly wake up crying and with vomiting of no apparent cause). This exam can be performed rapidly at practically all health stations with the use of glucose meters.

Urine :

Glycosuria and at times, ketonuria, are observed in moderate and serious cases.(42)(43)

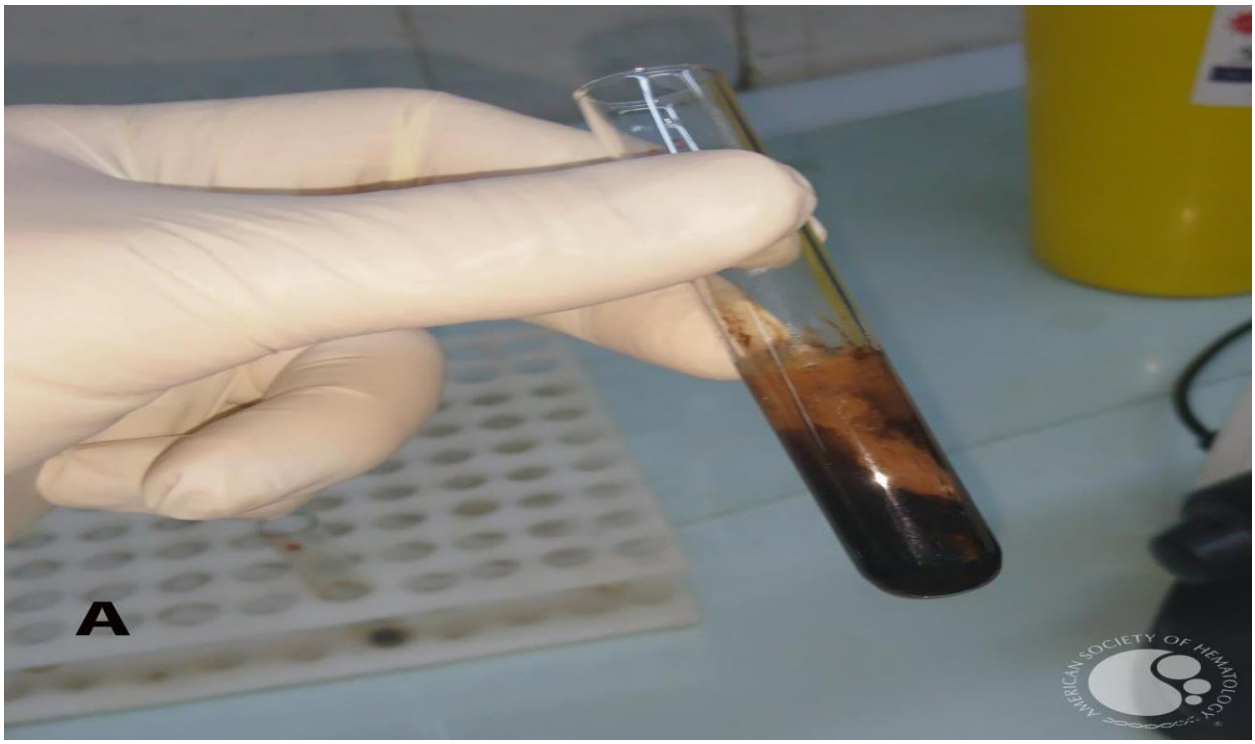


Fig 2.2 Image shows centrifuged urine sample of patient after addition of sulfosalicylic acid. Test was done to assess precipitation of protein in the urine sample. Presence of black color is due to severe hematuria.

Chest X-ray :

- May reveal an enlarged cardiac area and signs of APE, eventually unilateral.

- showing bilateral infiltrates suggested of pulmonary edema

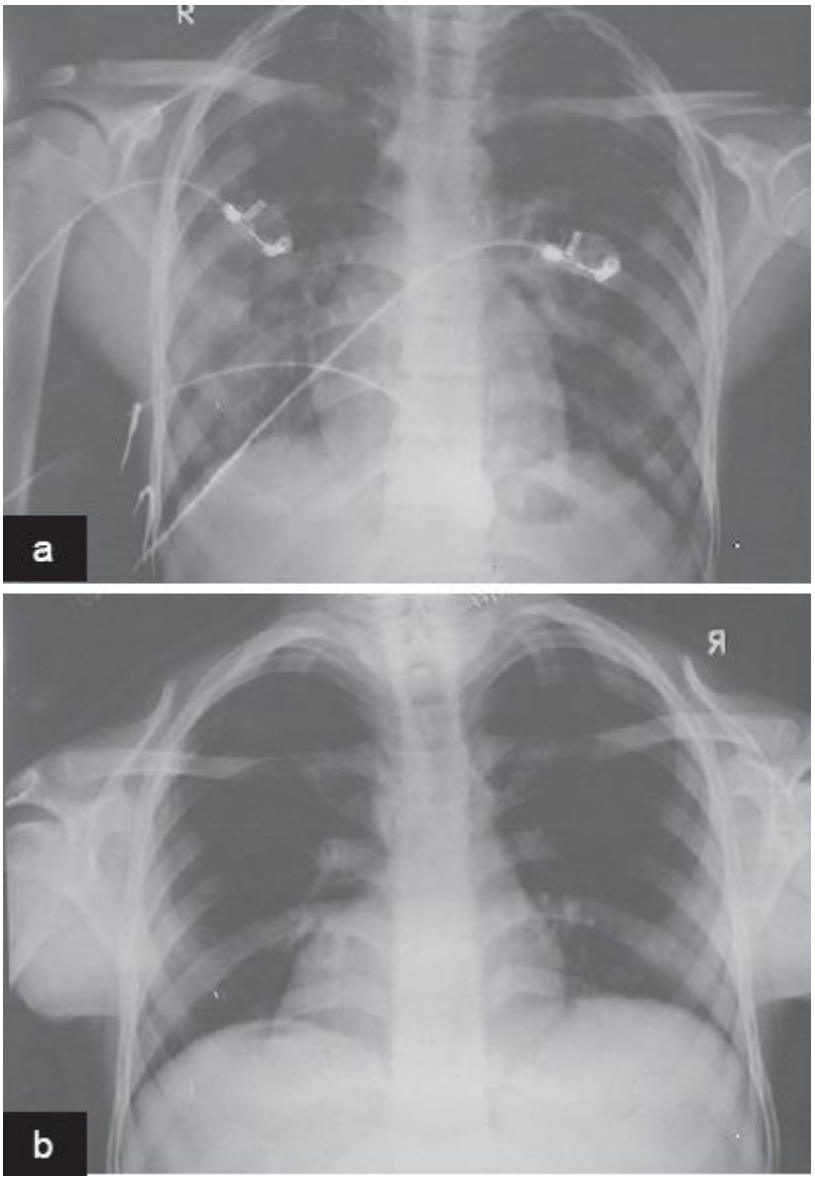
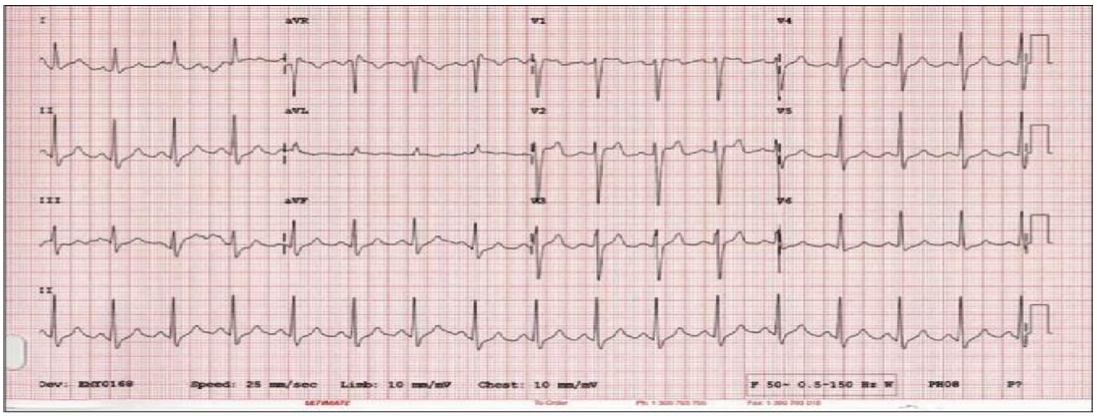


Fig 2.3X-ray of the patient showing bilateral infiltrates suggested of pulmonary edema (a). X-ray of the patient on day 3 showing bilateral infiltrates suggested of pulmonary edema (b). X-ray of the patient on day 5^{th} showing no infiltrates

Electrocardiologic and echocardiographic features of patients exposed to scorpion bite

The purpose of this study is to examine clinical progress and hemodynamic and electrocardiologic features (QT depression and heart rate variability [HRV]) of patients exposed to a scorpion bite. Seventeen patients bitten by scorpions, and, as a control group, 15 healthy subjects were included in the study. Standard electrocardiograph (ECG) records, 24-hour Holter-ECG, and Doppler echocardiographic examinations were performed. Holter ECG indicated sinus tachycardia, sinus bradycardia, paroxysmal supraventricular tachycardia, atrial fibrillation, first-degree and second-degree atrioventricular block not requiring treatment, early atrial beats, and early ventricular beats in the patients at frequencies of 82%, 12%, 35%, 12%, 8%, 70%, and 47%, respectively. HRV parameters that reflected parasympathetic activity (SD 35+/-13-43+/-16, RMS-SD: 20+/-9-30+/-12, high frequency: 7.8+/-2-4.3+/-3, $p<0.05$) were significantly lower ($p<0.05$). Low frequency, which especially showed sympathetic activity (LF: 11+/-13-11+/-23, $p>0.05$), was similar in both groups. In addition, the LF/HF ratio, which reflected sympathovagal

balance, was significantly increased in the patient group (1.5+/-1-3.0+/-2, p=0.005). Corrected QT and QT dispersion values were not significantly different with respect to the control (p>0.05). In the patient group compared to the control, a significant decrease was determined in the proportion of mitral E velocity to mitral A velocity (mEv/mAv), diastolic filling period (DFP), and left ventricular ejection fraction (LVEF), while a significant increase was noticed in pulmonary artery pressure (PAP) (mEv/mAv: 0.9+/-0.4-1.7+/-0.6, DFP: 362+/-8.5-425+/-89, LVEF: 53.1+/-6.7-68.6+/-5.8, PAP: 38.1+/-13-27.2+/-6, p<0.05). Scorpion bite leads to serious cardiovascular disorders, associated with decreased HRV, decreased systolic and diastolic functions, increased arrhythmic events, and hemodynamic disturbance with sympathetic and parasympathetic balance disturbance.(26)



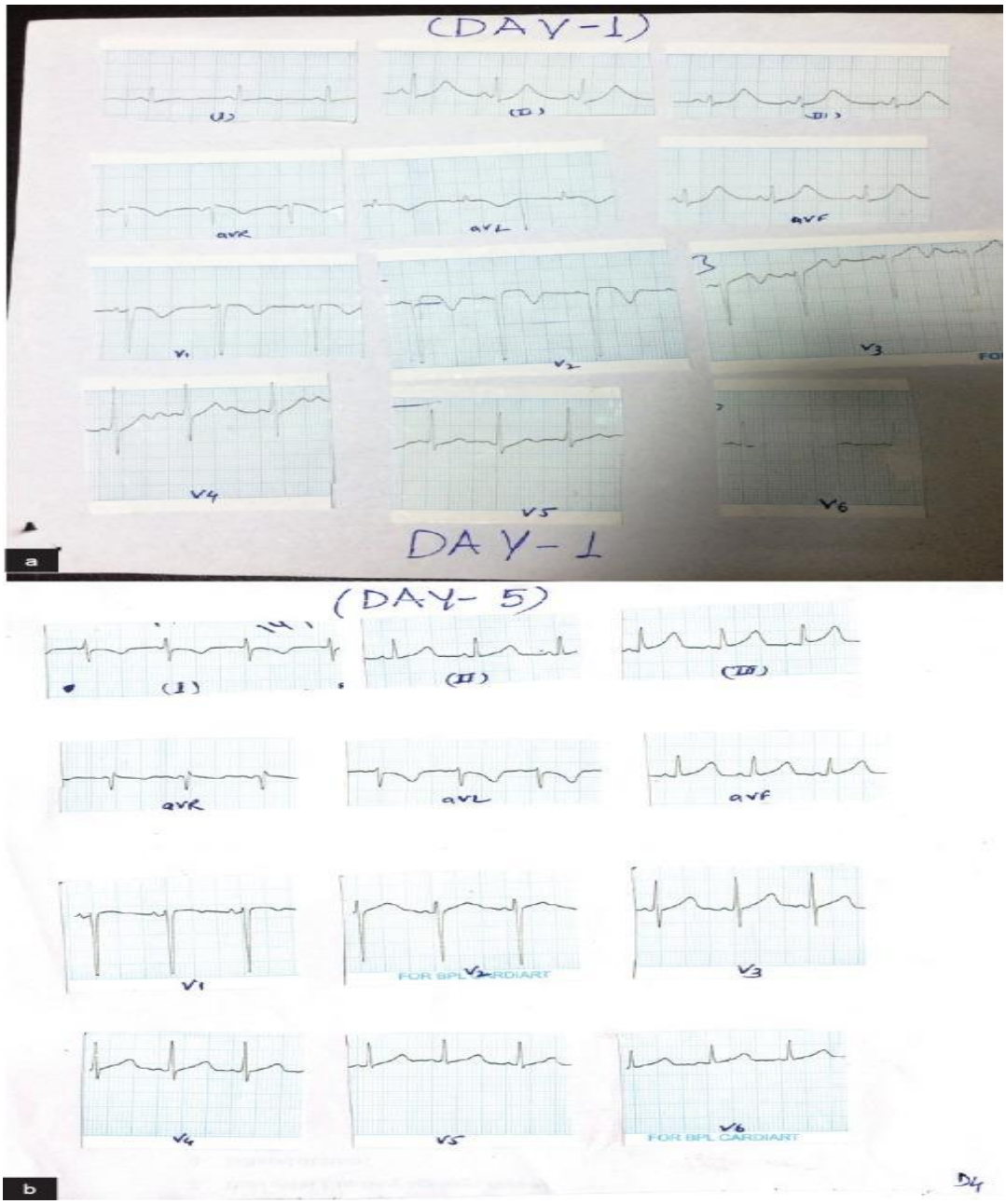
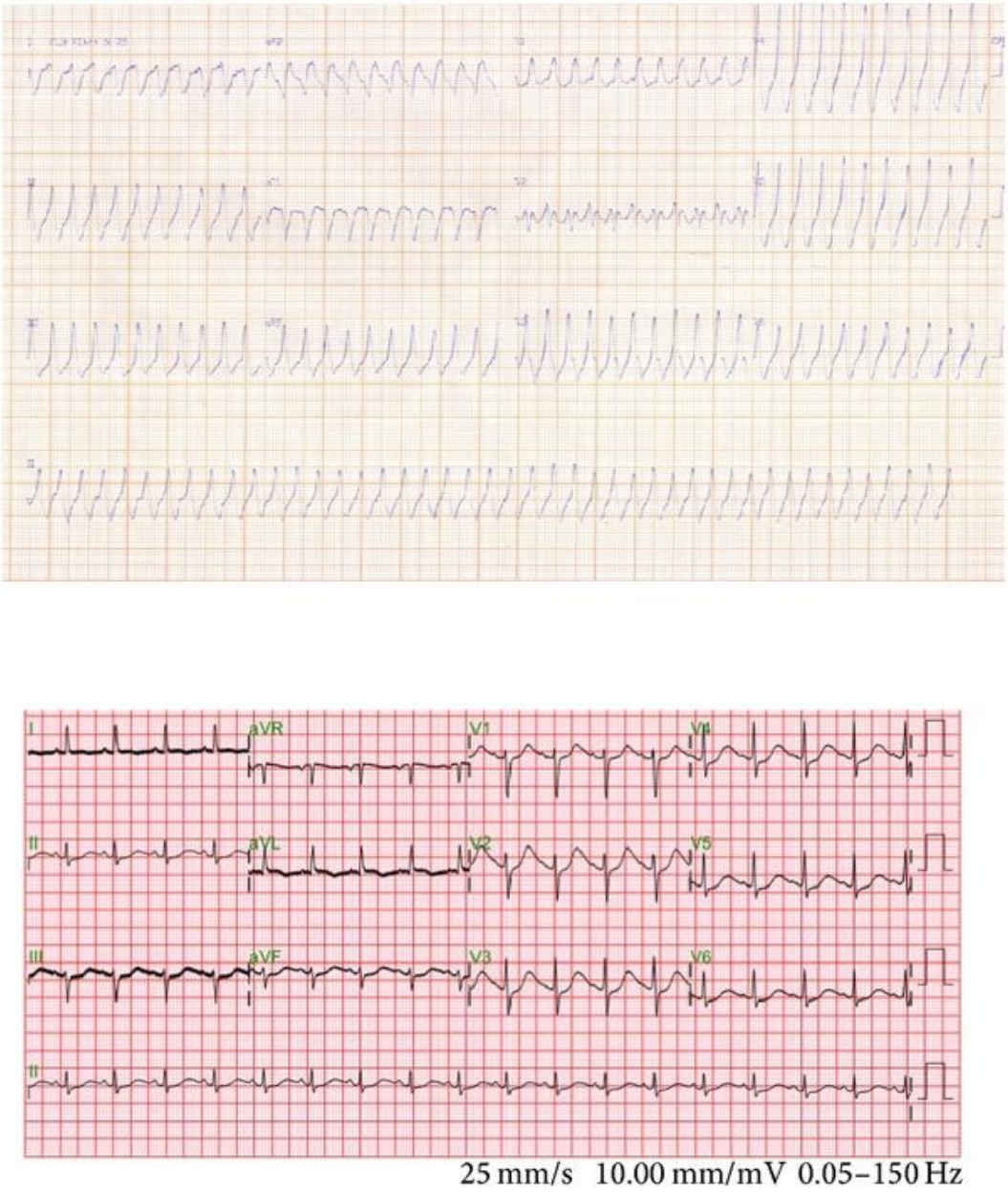


Fig 2.4 (a) Electrocardiogram (ECG) of the patient on the day of admission showing secondary ST- T changes and tachycardia, (B) ECG of the patient on day 5th showing T wave inversion in lead I and aVL



Electrocardiogram of a 25-year-old woman showing an episode of (a) sustained ventricular tachycardia (SVT) with very fast heart rate

(approximately 300 bpm) after scorpion envenomation (b). Upon admission to the hospital, the electrocardiogram showed sinus tachycardia associated with QTc interval prolongation (550 ms) after SVT reversal

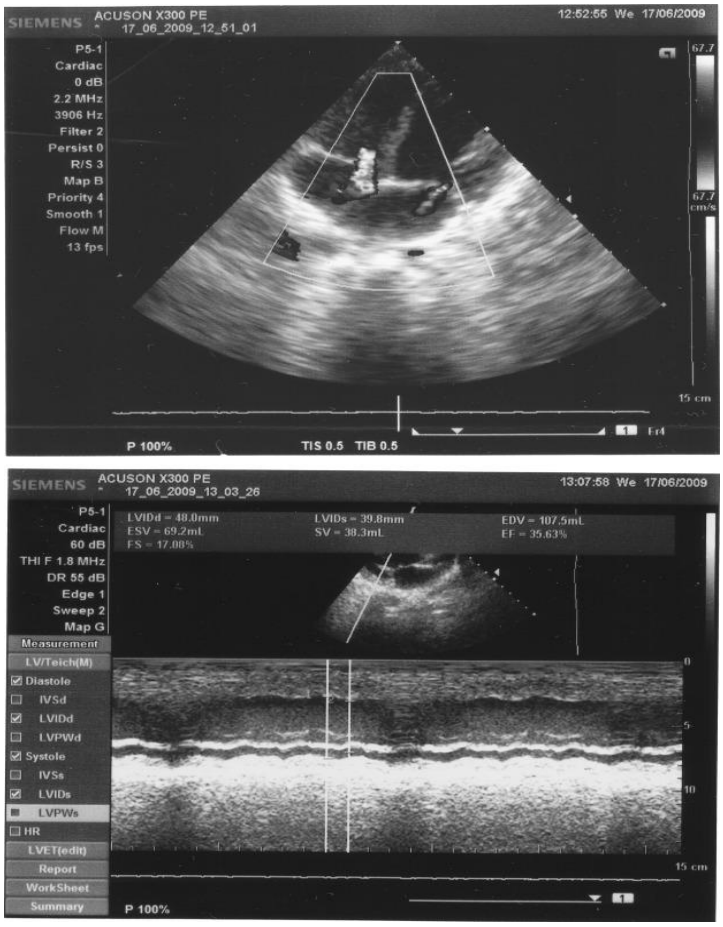
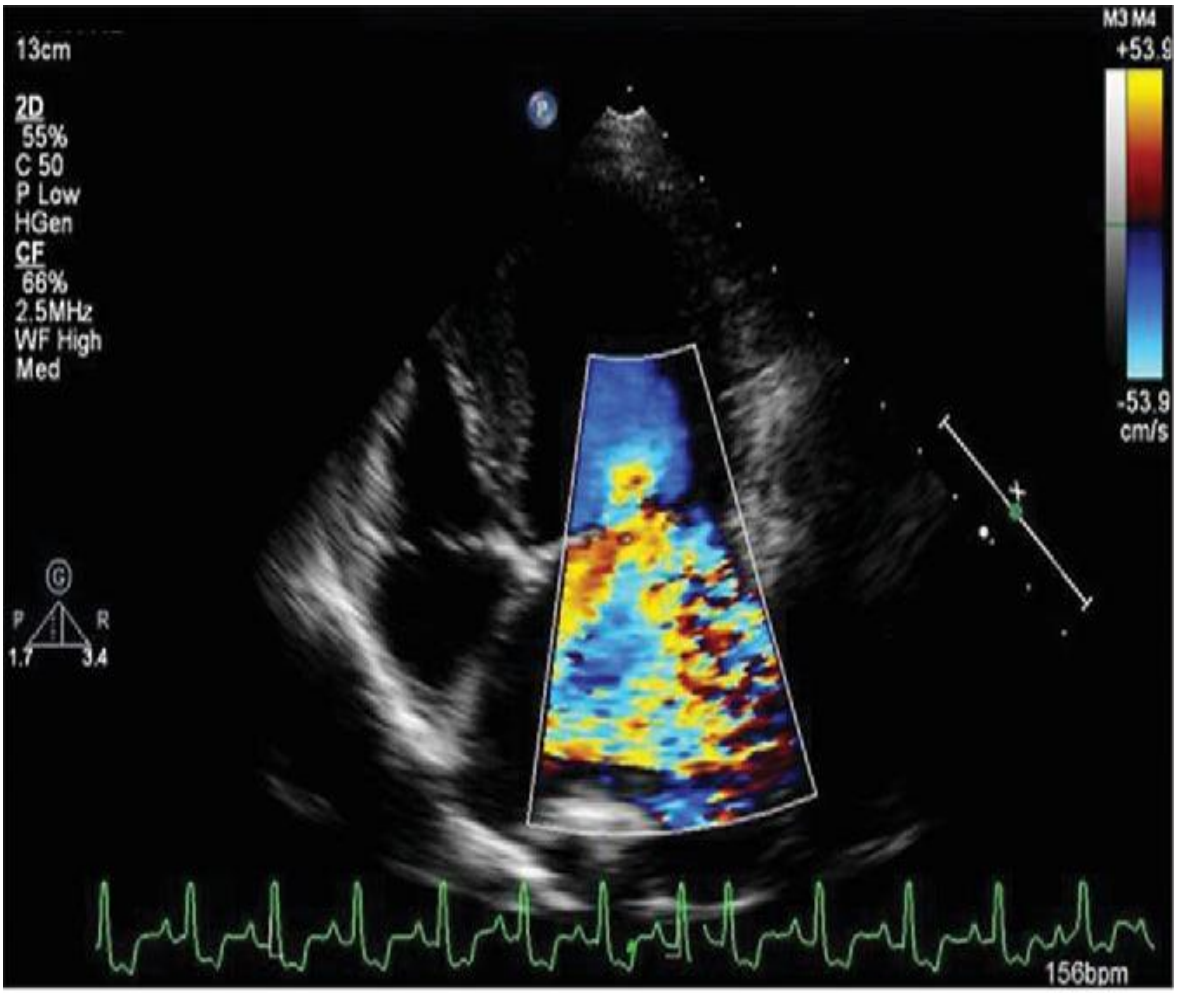


Fig. a. Regurgitation in Mitral and Tricuspid valves on Doppler echocardiography, b. Decreased FS and EF measurements on normal M-Mode echocardiography


13cm
2D
55%
C 50
P Low
HGen
CF
66%
2.5MHz
WF High
Med
M3 M4
+53.9
-53.9
cm/s
P
R
1.7
3.4
156bpm

Computed brain tomography :

May be useful when a cerebrovascular accident or other neurological complications are suspected.

PROGNOSIS

The prognosis is very good in cases of mild and moderate envenoming and in severe cases if properly treated. Some risk factors affect the prognosis, i.e., age of less than 10 years, scorpion size and species (T. serrulatus account for most serious cases of envenoming), and time between the sting and patient arrival at the hospital. In serious cases, SAV should be instituted rapidly as possible.The identification and treatment of clinical complications and early intubation, whenever it is necessary and considerably improves the prognosis of the patients, especially children. This highlights the importance of recognizing the severity of envenoming on the part of the professionals who provide first aid care, and who can count on the help of Toxicological Assistance Centers if doubt arise. It is also important that care to be taken in the initial treatment with saline expansion (risk of precipitating APE) and with the use of any other medication, even antiemetics. Analgesia should

be provided as soon as possible, oxygen saturation should be assessed and recorded, a venous access should be provided if needed, and the patient should be referred to a hospital where SAV is available.(40)

Treatment for scorpion bite:

Prazosin–a competitive post-synaptic alpha1, adreno-receptor antagonist–should be the first line of management, since alpha receptors stimulation plays a major role in the evoluation of clinical spectrum(9,58).

Prazosin suppresses sympathetic outflow and activates venom-inhibited potassium channels. It decreases the preload, afterload and blood pressure without increasing the heart rate. Prazosin counters vasoconstriction induced by endothelins through accumulation of cyclic GMP (cGMP). Prazosin by inhibiting phospho-diesterase enzyme and by inhibiting the formation of inositol triphosphate makes this possible. cGMP, a second messenger of nitric oxide in vascular endothelium (eNOS) and myocardium prevents further myocardial injury. The metabolic and hormonal effects of alpha receptors stimulation are

reversed by prazosin. Thus prazosin is a cellular and pharmacologic antidote to the actions of scorpion venom and it is also cardioprotective.

Prazosin is available as scored 1 mg tablet. Sustained release tablets are not recommended in this condition. The dose recommended is 30 microgram/kg/dose. This is given as an immediate measure in all with evidence of autonomic storm. It should not be given as prophylaxis in children when pain is the only symptom. In case of vomiting, it can be administered through nasogastric tube. After giving prazosin, mother should be advised not to lift the child to prevent the effects of 'First dose phenomenon' due to prazosin. Oral hydration and milk feeds must be encouraged. If needed, intravenous maintenance fluids should be given to correct dehydration due to excessive sweating and vomiting. Prazosin can be given irrespective of blood pressure provided there is no hypovolemia. Blood pressure, pulse rate and respiration must be monitored every 30 minutes for 3 hours, every hour for next 6 hours and later every 4 hours till improvement. Prazosin should be repeated in the same dose at the end of 3 hours according to clinical response and later every 6 hours till extremities are warm, dry and peripheral veins are

visible easily. The time lapse between the sting and administration of prazosin for symptoms of autonomic storm determines the outcome(53,58). No more than four doses have been required in majority of children treated at our center.

Benzodiazepines (Diazepam) is often useful to quieten a child restless after scorpion sting. Benzodiazepines in concert with GABA open chloride ion channel. This effect of diazepam antagonises the scorpion toxins' ability to stimulate specific ion channel.

Pain and Fluid Management

Pain relief is useful since it allays anxiety and avoids myocardial stress. However, many children have only mild and tolerable pain; when severe, NSAIDS provide prolonged relief. Local ice packs, xylocaine (local anesthetic), dehydroemetine (counter irritant) and streptomycin (neuromuscular blockade) have been reported to be useful(3,49,50).

The loss of fluid due to profuse sweating and vomiting is usually overlooked. So oral fluids whenever feasible must be encouraged. When children present with tachypnea and altered sensorium, parenteral fluids

(N/5 saline) are required. Fluid requirements need to be balanced carefully. In children with pulmonary edema, CVP monitoring is essential.

The use of a combination of insulin and alphablocker with NaHCO3 resulted in reversal of all electrocardiographic changes (rhythm disturbances, conduction defects, ischemia and infarction like pattern) to sinus rhythm in experimental animals(51). Bawaskar reported similar changes with oral prazosin in his patients(48).

Treatment of Pulmonary Edema

Pulmonary edema in these children is mainly due to myocardial dysfunction. Though serious in itself, it does not necessarily mean a poor prognosis. Despite diagnostic and therapeutic advances in medicine, treatment of myocardial dysfunction is primarily supportive.

In children with pulmonary edema with or without hypertension, management should be directed towards relieving afterload without compromising preload. The use of diuretics to minimize or reduce fluid overload seems a reasonable measure but only when renal water

excretion is impaired. Otherwise the best way to prevent fluid overload is to maintain an adequate cardiac output. Thus dobutamine support (5-15 mg/kg/min) with vasodilatation through sodium nitroprusside (0.3-5 mg/kg/min) or nitroglycerine (5 mg/min) infusate is preferred in this situation. Prazosin is to be given one hour before termination of sodium nitroprusside (SNP) drip. If SNP is not available, one can use isosorbide dinitrate 10 mg every 10 minutes sublingually as an emergency measure. Morphine, a standard therapy in pulmonary edema, should be avoided in scorpion sting, since narcotics worsen dysrythmias in these children.

Occasionally, children with scorpion sting present with multi-organ failure. A systemic inflammatory response is presumably the cause; however our knowledge on the pathogenesis of such a state is still incomplete. Presence of respiratory failure with or without CNS disturbances in the presence of hypertension or complicating those children with pulmonary edema should be aggressively treated with early ventilation, afterload reduction, careful sedation and acid-base correction.

Scorpion Antivenom

Scorpion venoms reach their target too rapidly to be neutralized and anti-venom within 30 minutes of sting may reverse their effect. Usefullness of scorpion anti-venom varies between countries. Doctors from Brazil, Mexico and Saudi Arabia report benefit(52–54). Systematic administration of scorpion antivenin did not alter the clinical course of scorpion sting in a matched pair study undertaken at an intensive care unit in Tunisia(55). Antivenom against the toxins of Indian scorpions is not available for clinical use. Moreover children reach hospital late already exhibiting cardiac manifestations. It is not clear from published reports whether antivenom is effective in prevention or abolition of cardiovascular manifestations. It would be practical to neutralize the effects of an overstimulated autonomic nervous system through prazosin than attempting to neutralize toxin already bound to receptors on sodium channel.

Unhelpful Treatment

Standard therapy was not clearly defined in earlier days; many therapies were in vogue without experimental justification.

- *Lytic Cocktail* (Pethidine + Promethazine + Chlorpromazine): The alpha blocking effect of chlorpromazine might be beneficial; but pethidine may convert sublethal dose of scorpion venom into a lethal one and they also interfere with protective respiratory reflexes(3,12). We no longer use lytic cocktail at our center.
- *Morphine*: worsens dysrythmias(4).
- *Steroids*: In 600 consecutive patients of scorpion sting randomly assigned to receive hydrocortisone and placebo, no significant difference was found in steroids and placebo groups(56). Moreover steroids might enhance the necrotizing effects of excessive catecholamines on myocardium(10).
- *Atropine*: Complete abolition of parasympathetic effects may permit the domination of the overstimulated sym-pathetic system. Atropine potentiates tachycardia and sustains hypertension(57).

- *Nifidepine*: Reflex tachycardia and negative inotropic effect argues against its use(8); despite its antihypertensive and vasodilator effect, 35% of scorpion victims developed myocardial failure and 14% acute pulmonary edema(48,58,59).
- *Ace Inhibitors*: (Captopril) aggrevate hyperkalemia and inhibit breakdown of bradykinin, which is implicated in experimental pulmonary edema due to scorpion sting. Captopril failed to correct hemodynamics in two cases and did not prevent cardiac arrythmias(60

MATERIALS AND METHODS

SOURCE OF DATA:

All patients hospitalized for scorpion sting in Tirunelveli medical college during study period

Study type:

Hospital based prospective study

Sample size and study period:

All the patients admitted for scorpion envenomation during the study period april 2021 to September 2022

Inclusion criteria:

Patients between the age group of 13-70 years with definite history of scorpion bite

Patients admitted within first 24 hours of scorpion bite

Exclusion criteria:

The patient with doubtful scorpion bite

Patient with known heart disease(congenital and acquired)

Patient with recent trauma or surgical intervention

Pediatric age groups and patient more than 70 years of age.

OBSERVATION AND RESULTS

Table 1 and figure 1: age distribution of cases

Age Group	No of cases	Percentage
<20	6	10.00%
20-29	14	23.33%
30-39	10	16.67%
40-49	12	20.00%
50-59	10	16.67%
60-69	5	8.33%
70-80	3	5.00%
Grand Total	**60**	**100.00%**
Mean	**39.95**	
SD	**16.1449**	

regarding age distribution of cases maximum nuber of cases 23% comes in the age group of 20-29 years of age.Mean age is 39.95 years and standard deviation is 16.1449

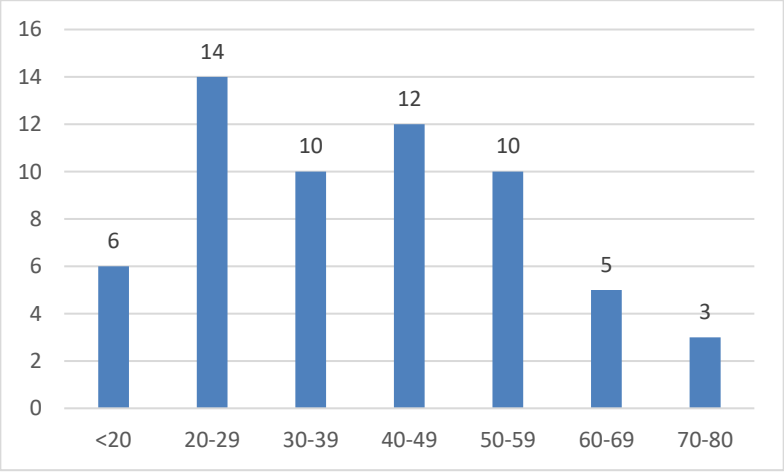


Figure 1:age distribution of cases

Table 2:sex distribution of cases

Gender	No of cases	Percentage
Male	40	66.67%
Female	20	33.33%
Grand Total	**60**	**100.00%**

Among 60 cases 66% of cases belongs to male gender and 20% of cases belongs to female gender.

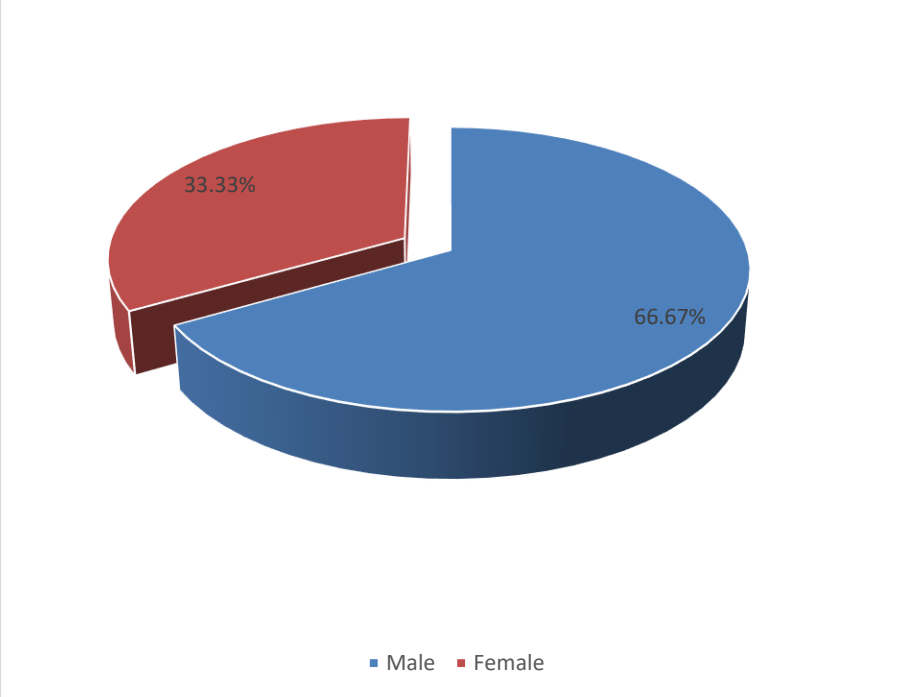


Figure 2: sex distribution of cases

Table 3:age group with gender distribution

Age Group	Gender					
	Male		Female		Total	
	No of cases	Percentage	No of cases	Percentage	No of cases	Percentage
<20	5	8.33%	1	1.67%	6	10.00%
20-29	10	16.67%	4	6.67%	14	23.33%
30-39	7	11.67%	3	5.00%	10	16.67%
40-49	7	11.67%	5	8.33%	12	20.00%
50-59	6	10.00%	4	6.67%	10	16.67%
60-69	2	3.33%	3	5.00%	5	8.33%
70-80	3	5.00%	0	0.00%	3	5.00%
Grand Total	**40**	**66.67%**	**20**	**33.33%**	**60**	**100.00%**
Mean	**38.975**		**41.9**		**39.95**	
SD	**16.8606**		**14.8285**		**16.1449**	
p =0.59						

Among the male gender maximum number of patients belongs to 20-29 years of age 16%.among female maximum number of cases belongs to age group 40-49 years of age.mean age for male sex is 38.975 and standard deviation was found to be 16.8606.mean age for female gender is 41.9 and standard deviation was 14.8285.The p value was found to be 0.59 and it is statistically insignificant.

Figure 3: age group with gender distribution

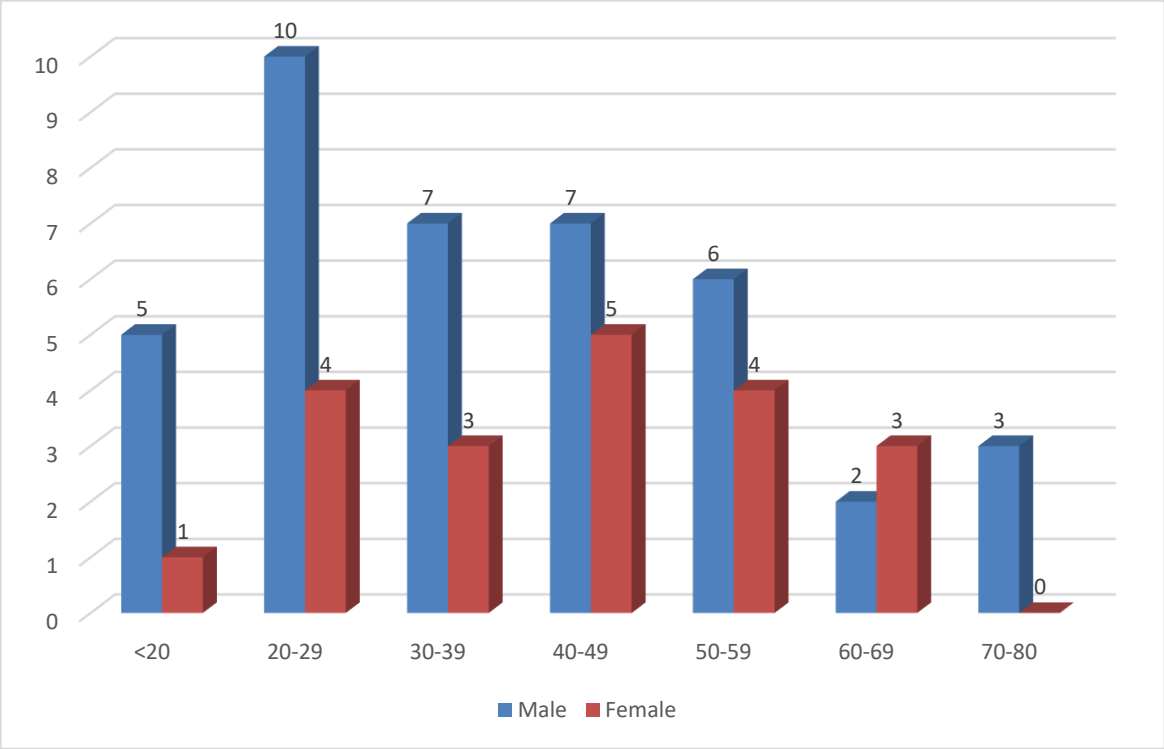


Table 4:distribution of cases according to symptoms :

Symptoms	**No of cases**
Local pain	37
Local swelling	26
Paraesthesia	15
chest pain	4
Sweating	11
Palpitation	7
Breathlessness	11
Giddiness	4
Redness at sting site	8
Vomiting	2

Regarding distribution of cases according to symptoms majority of cases had local pain.

Figure4: distribution of cases according to symptoms

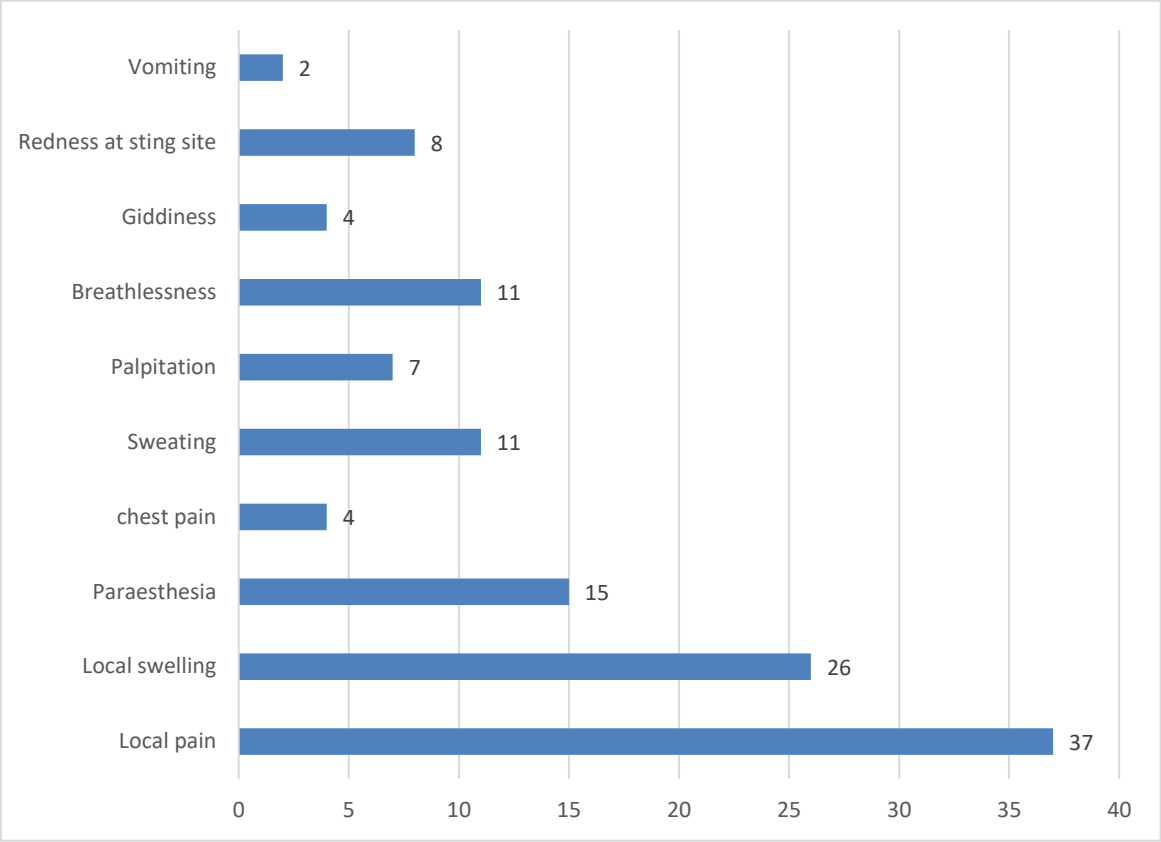


Table 5:distribution of cases according to signs

Signs	**No of cases**
Tachycardia	22
Hypotension	17
Hypertension	2
Profuse sweating	11
Cold peripheries	5
Tenderness at sting site	22
Pulmonary edema	3

Regarding distribution of cases according to signs majority of cases had tachycardia and cold peripheries and minimum number of cases has hypertension.

Figure 5:distribution of cases according to signs

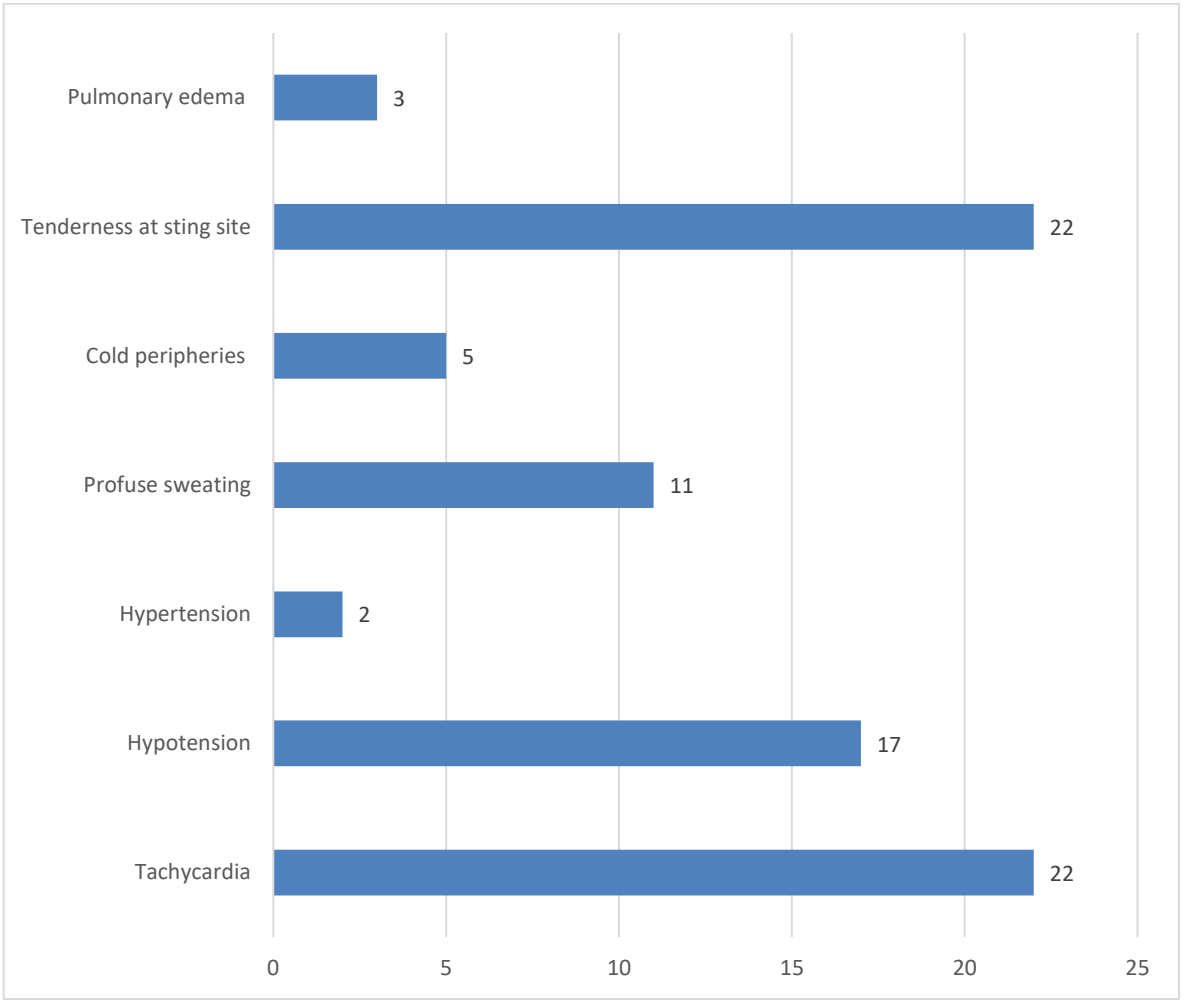
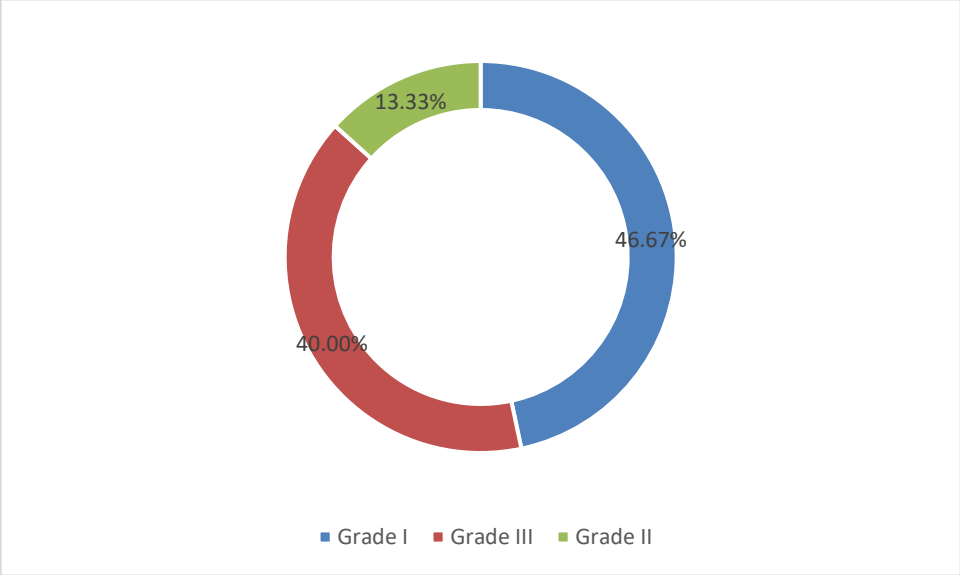

Pulmonary edema
3
Tenderness at sting site
22
Cold peripheries
5
Profuse sweating
11
Hypertension
2
Hypotension
17
Tachycardia
22
0
5
10
15
20
25

Table 5 and figure 5:distribution of cases according to grade:

Grade	**No of cases**	Percentage
Grade I	28	46.67%
Grade III	24	40.00%
Grade II	8	13.33%
Grand Total	**60**	**100.00%**



Regarding distribution of cases according to grade,46% of cases are grade 1 ,40 % of cases are grade 2 and 13% of cases are grade 3.

Table 6 :distribution of cases according to ECG changes:

ECG	**No of cases**	Percentage
Sinus Tachycardia	11	18.33%
Sinus Tachycardia with ST depression	4	6.67%
ST Elevation	2	3.33%
ST Depression	2	3.33%
ST Depression with T wave inversion	1	1.67%
Right bundle branch block	1	1.67%
Tall ‘T’ waves	3	5.00%
Complete heart block	1	1.67%
Left anterior fascicular block	2	3.33%
Grand Total	**27**	45.00%

Regarding distribution of cases according to ECG changes45% of cases show ecg changes among that.sinus tachycardia seen in 11 cases,sinus tachycardia with ST depression seen in 4 cases.ST elevation seen in 2 cases,ST depression seen in 2 cases,ST depression with T wave inversion seen 1 case,right bundle branch block seen in 1 case,Tall T waves seen in

3 cases,complete heart block seen in 1 case and left bundle fascicular block seen in 2 cases.

Figure6:distribution of cases according to ECG changes:

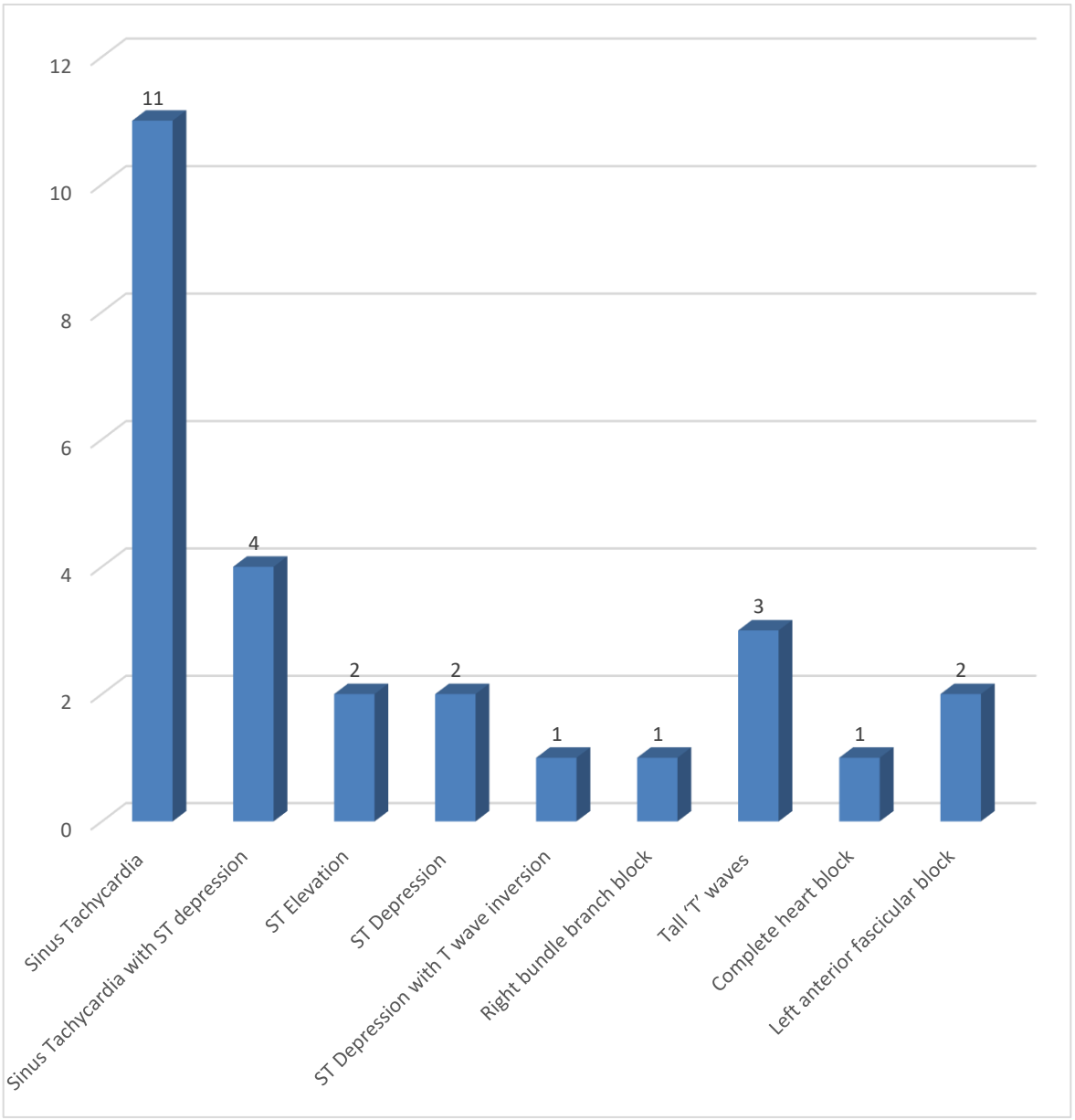


Table 7:distribution of cases according to RBS values.

RBS	**No of cases**	Percentage
<200	55	91.67%
>200	5	8.33%
Grand Total	**60**	**100.00%**

Regarding distribution of cases according to RBS values,91% of cases had blood sugar less than 200 and 8% of cases had blood sugar more than 200.

Figure 7: distribution of cases according to RBS values.

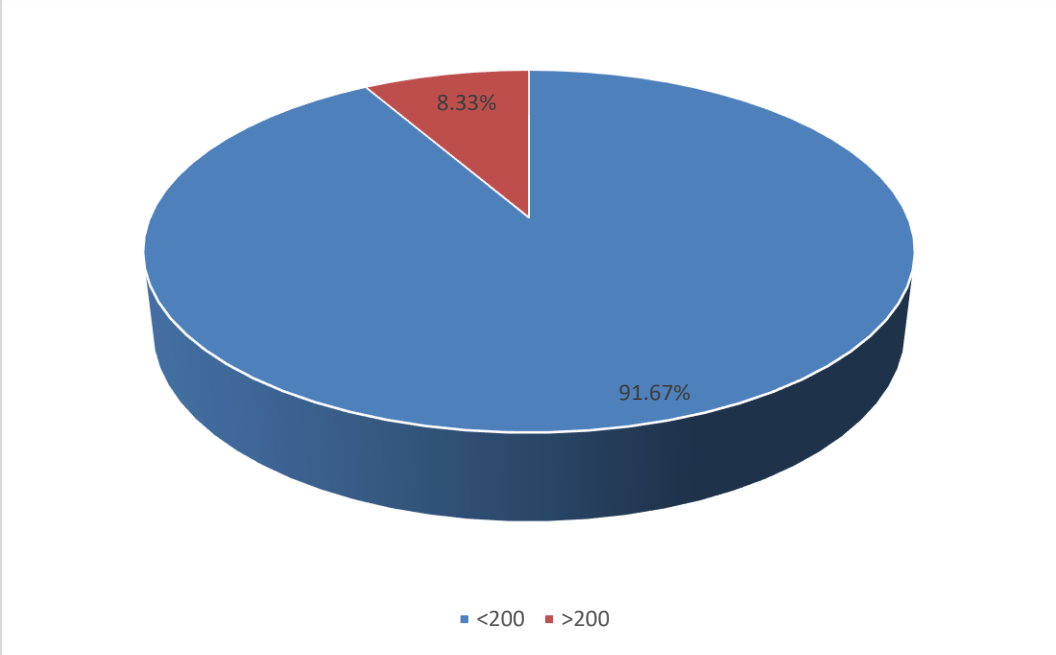


Table 8:distribution of cases according to time taken to reach hospital:

Time in Hours	**No of cases**	Percentage
<1	3	5.00%
1-3	36	60.00%
3-5	19	31.67%
>5	2	3.33%
Grand Total	**60**	**100.00%**

Regarding distribution of cases according to time taken for reaching the hospital.3 cases reached with in less than 1 hour,36 cases reached in 1-3 hours,19 cases reached in 3-5 hours and 2 cases reached the hospital after 5 hours.

Figure:8: distribution of cases according to time taken to reach hospital:

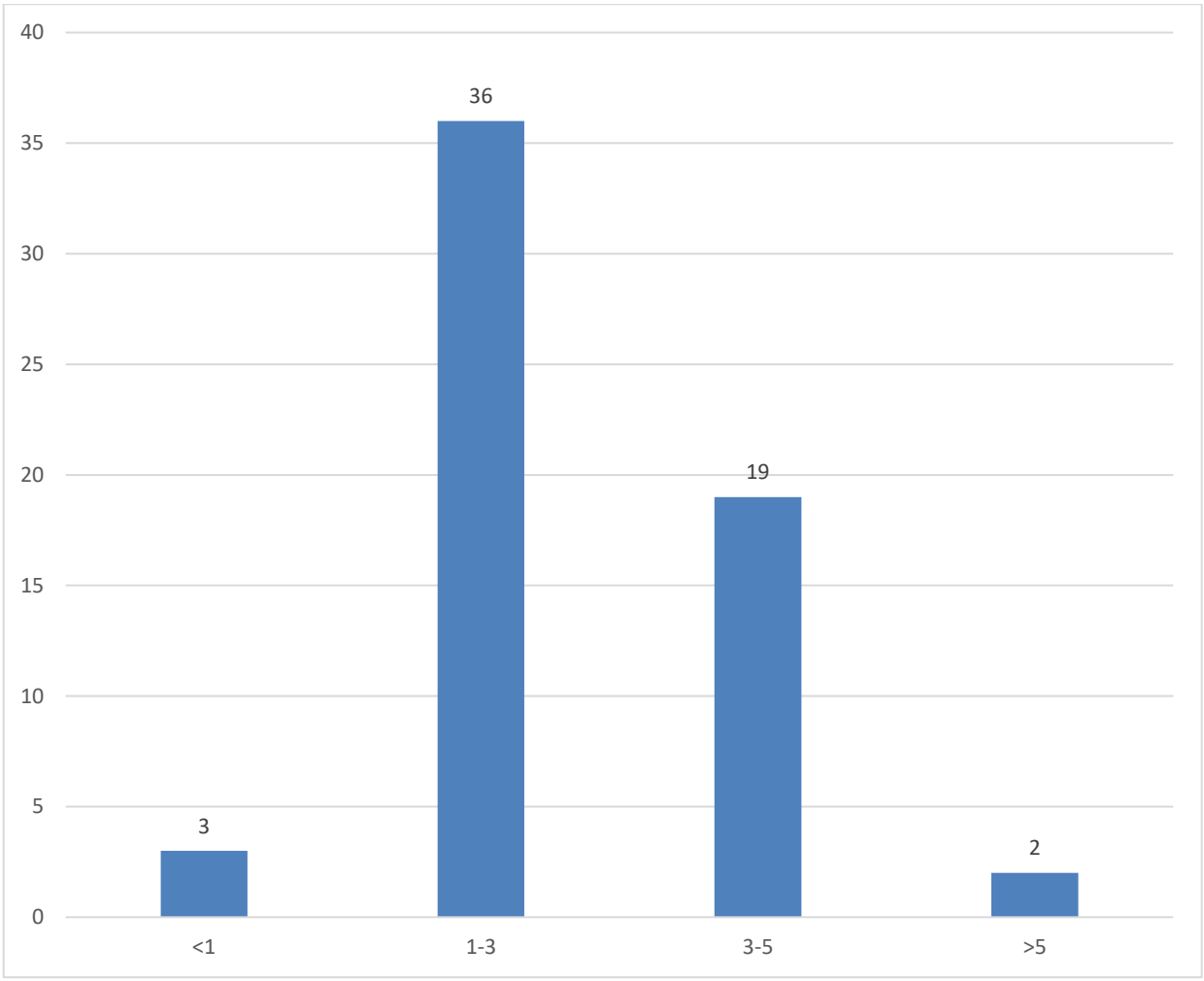
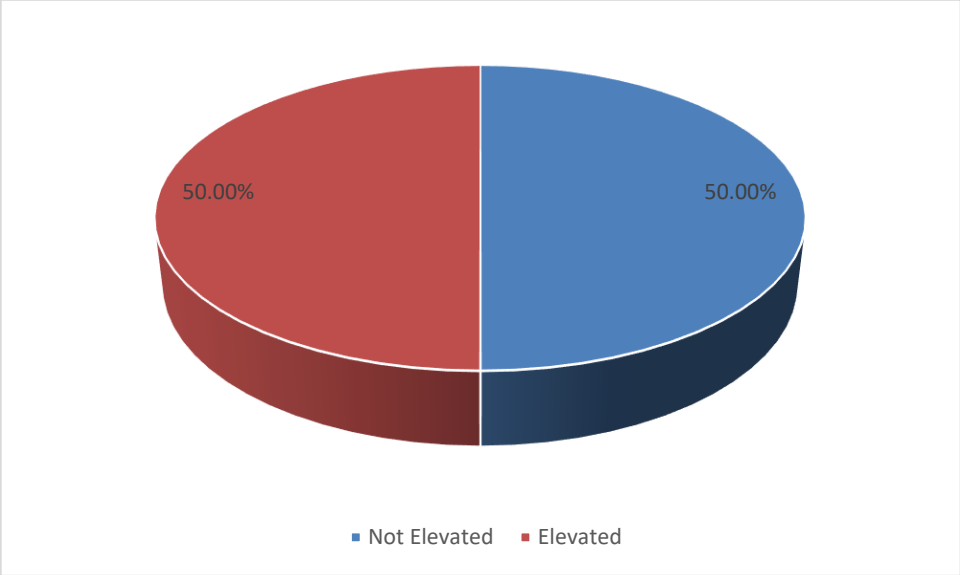


Table 9 and figure9: distribution of cases according to CPK MB level.

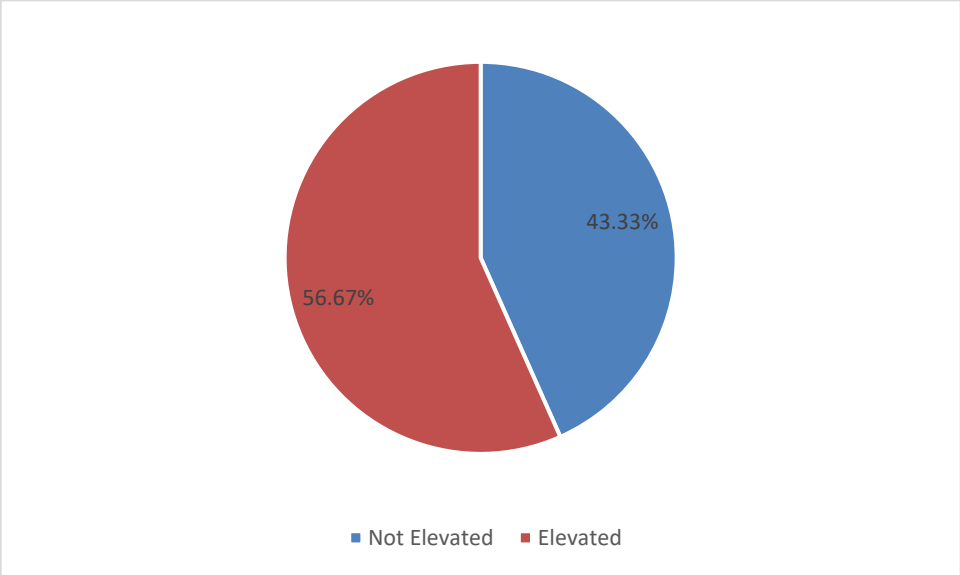
CPK MB	**No of cases**	Percentage
Not Elevated	30	50.00%
Elevated	30	50.00%
Grand Total	**60**	**100.00%**



Out of 60 cases studied,30 cases had elevated CPK MB and 30 cases CPK MB not elevated.

Table 10 and figure 10:distribution of cases according to SGOT level.

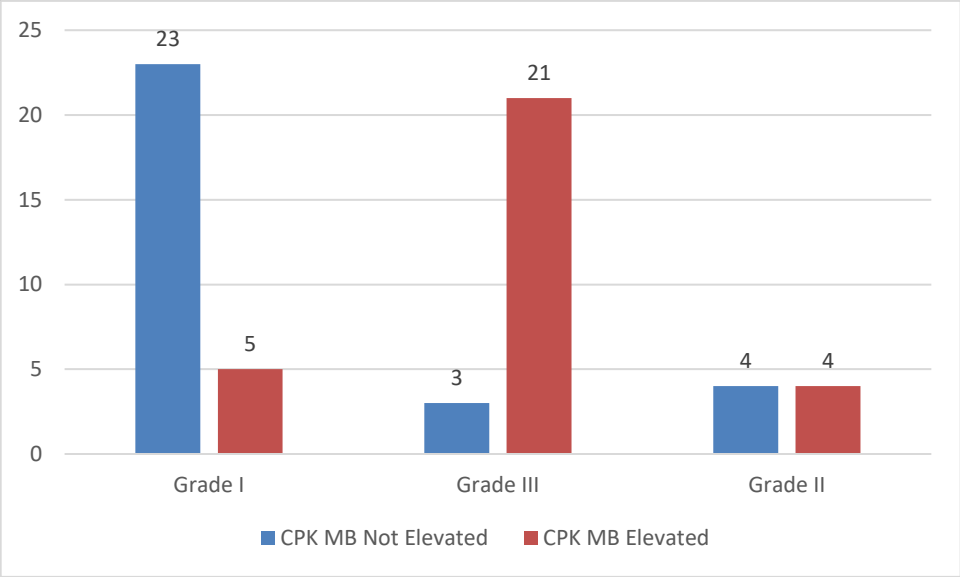
SGOT	**No of cases**	Percentage
Not Elevated	26	43.33%
Elevated	34	56.67%
Grand Total	**60**	**100.00%**



Out of 60 cases studied 34 cases SGOT got elevated and in 26 cases SGOT not elevated.

Table 11 and figure 11:grade of sting vs CPK MB level

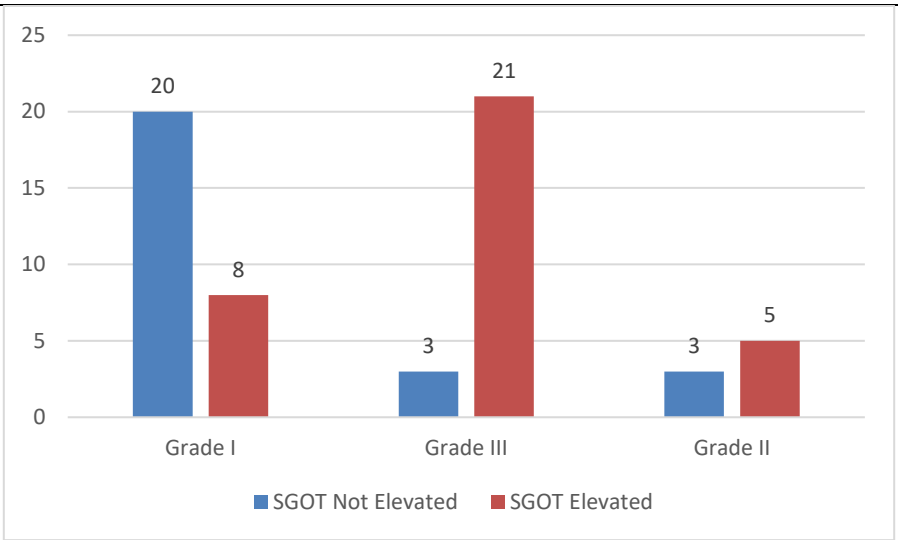
Grade	CPK MB		Grand Total
	Not Elevated	Elevated	
Grade I	23	5	28
Grade III	3	21	24
Grade II	4	4	8
Grand Total	**30**	**30**	**60**
p = 0.000003			



CPK MB level is elevated in grade 3 sting and p value was found to be 0.00003 and it is statistically significant.

Table 12 and figure 12:grade of sting with SGOT levels

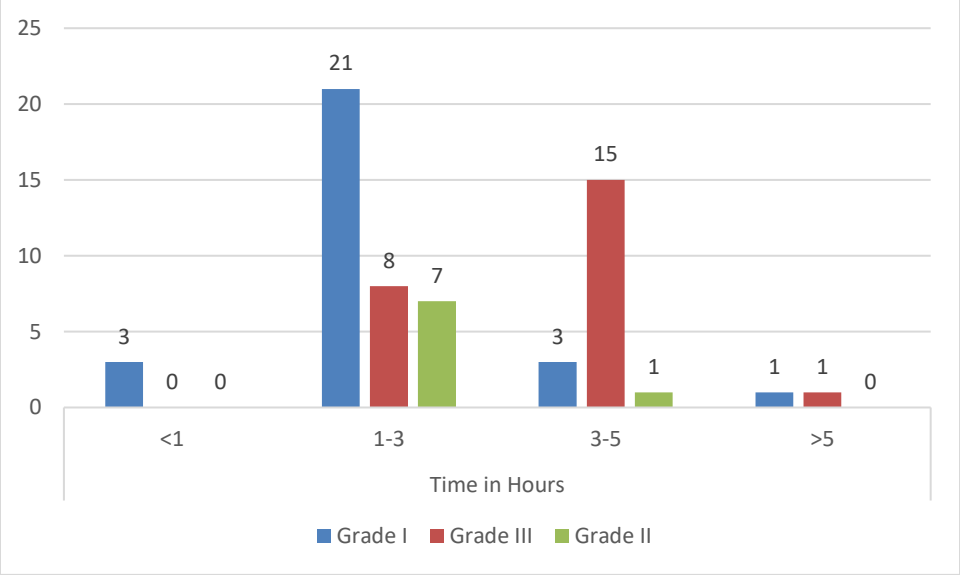
Grade	SGOT		Grand Total
	Not Elevated	Elevated	
Grade I	20	8	28
Grade III	3	21	24
Grade II	3	5	8
Grand Total	**26**	**34**	**60**
p=0.0001			



Regarding statistical test between grade of sting and SGOT levels.out of 34 cases having increased SGOT levels 21 cases had grade 3 injuries and p value was found to be 0.0001 and it is statistically significant.

Table 13 and figure 13:Grade vs time in hours:

Grade	Time in Hours				Grand Total
	<1	**1-3**	**3-5**	**>5**	
Grade I	3	21	3	1	28
Grade III	0	8	15	1	24
Grade II	0	7	1	0	8
Grand Total	**3**	**36**	**19**	**2**	**60**
p=0.002					



Regarding grade of injury and time in hours ,statistical test was done and p value was found to be 0.002 and it is statistically significant.

Table 14:Time in hours vs ECG changes

Time in Hours	ECG		Grand Total
	Normal	**Abnormal**	**Grand Total**
<1	2	1	3
1-3	26	10	36
3-5	4	15	19
>5	1	1	2
Grand Total	**33**	**27**	**60**
p=0.003			

Out of 60 cases 27 cases developed abnormal ECG findings and the p value was found to be 0.003.Statistically significant p value was obtained.

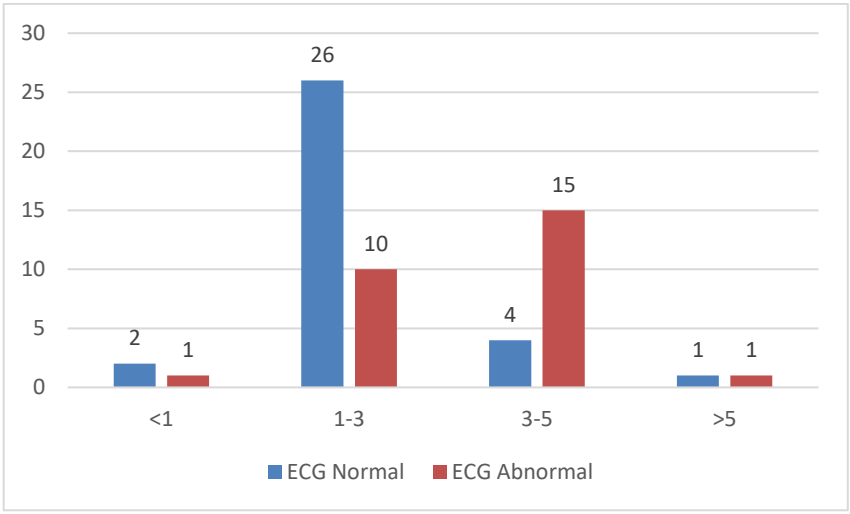
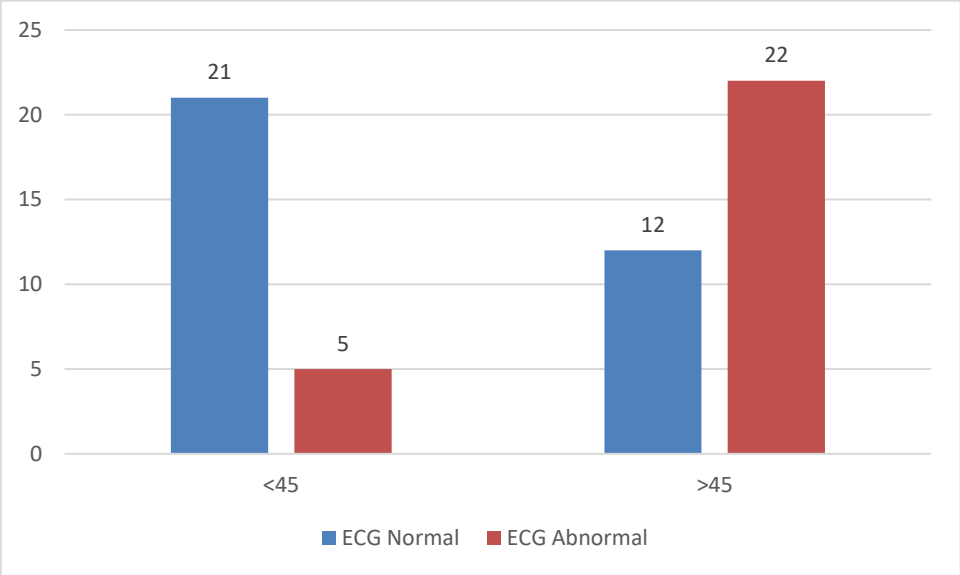


FIGURE 14: Time in hours vs ECG changes

Table 15 and figure 15:SGOT vs ECG

SGOT	ECG		Grand Total
	Normal	**Abnormal**	
<45	21	5	26
>45	12	22	34
Grand Total	**33**	**27**	**60**
p=0.0004			



Out of 34 cases with elevated SGOT values 22 cases developed abnormal ECG and p value was found to be 0.00004 and it is statistically significant.

Table 16 and figure 16:CPK MB values with ECG

CPK MB	ECG		Grand Total
	Normal	**Abnormal**	
<25	25	5	30
>25	8	22	30
Grand Total	**33**	**27**	**60**
p=0.00001			

Out of 30 cases with elevated CPK MB values 22 cases developed abnormal ECG and p value was found to be 0.00001 and it is statistically significant.

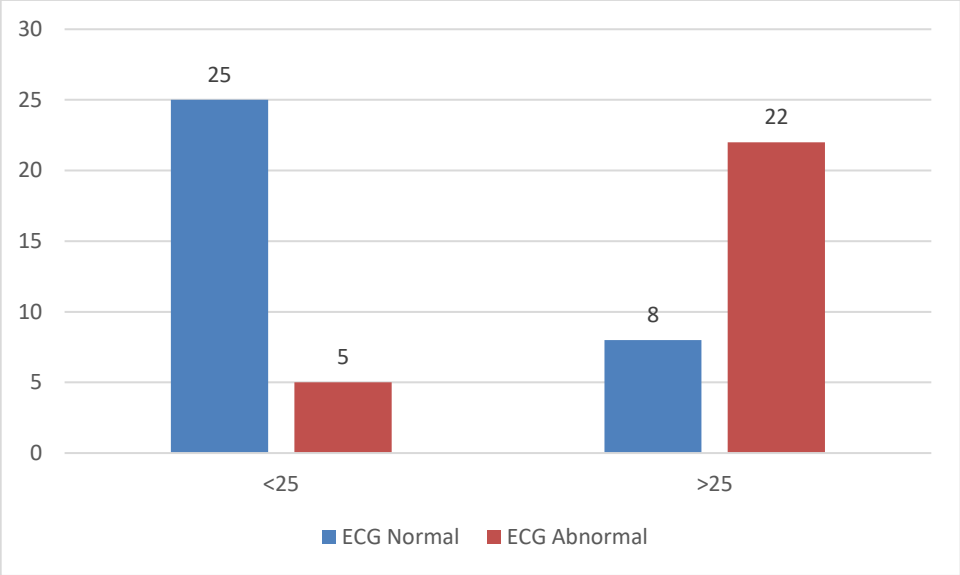
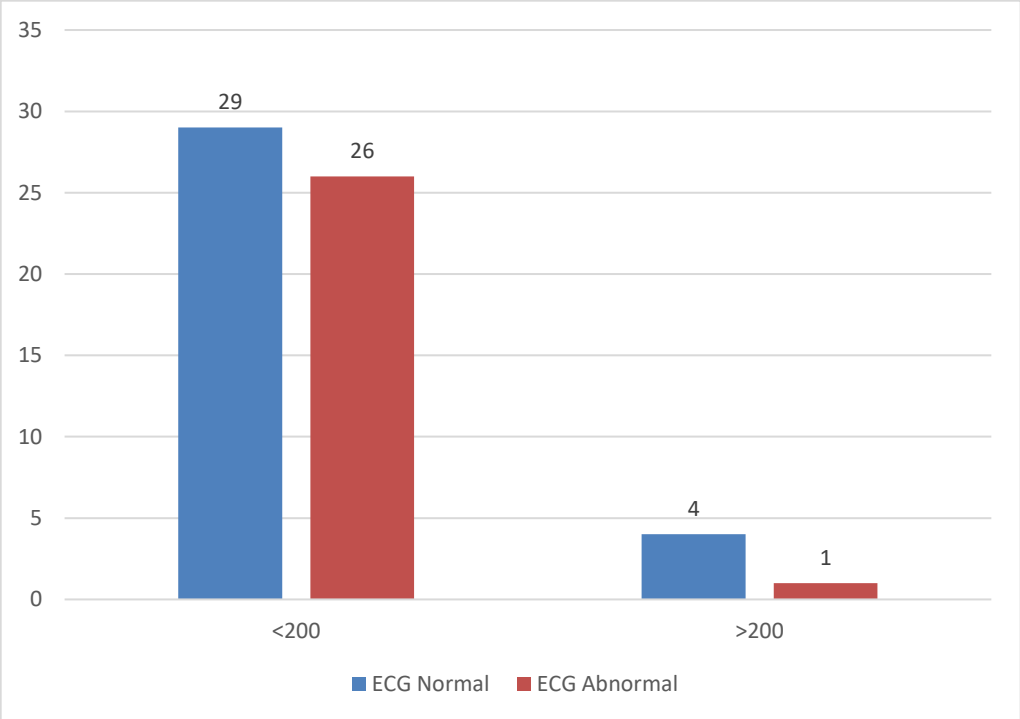


Table 17 and figure 17:RBS with ECG

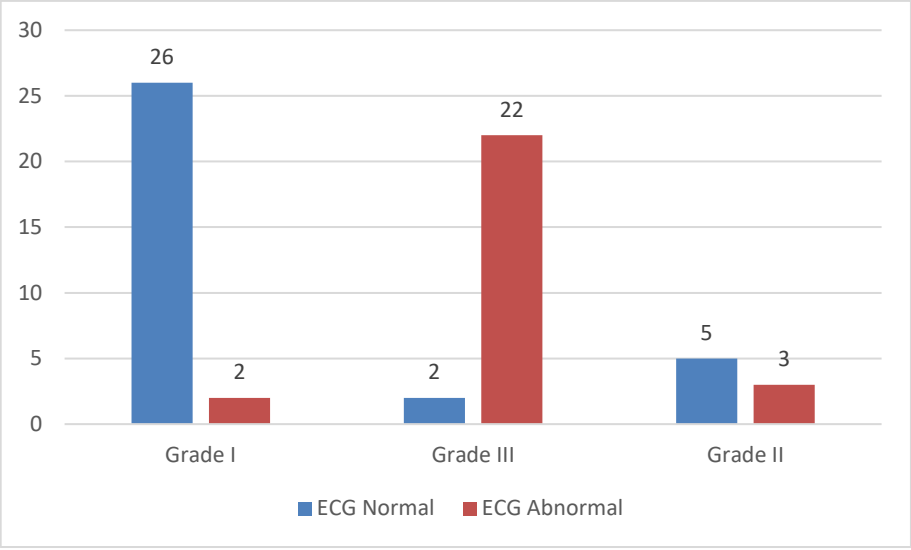
RBS	ECG		Grand Total
	Normal	**Abnormal**	
<200	29	26	55
>200	4	1	5
Grand Total	**33**	**27**	**60**
p=0.24			



When RBS with ECG changes p value was found to be 0.24 and it is statistically insignificant.

Table 18 and figure 18:grade of sting with ECG changes

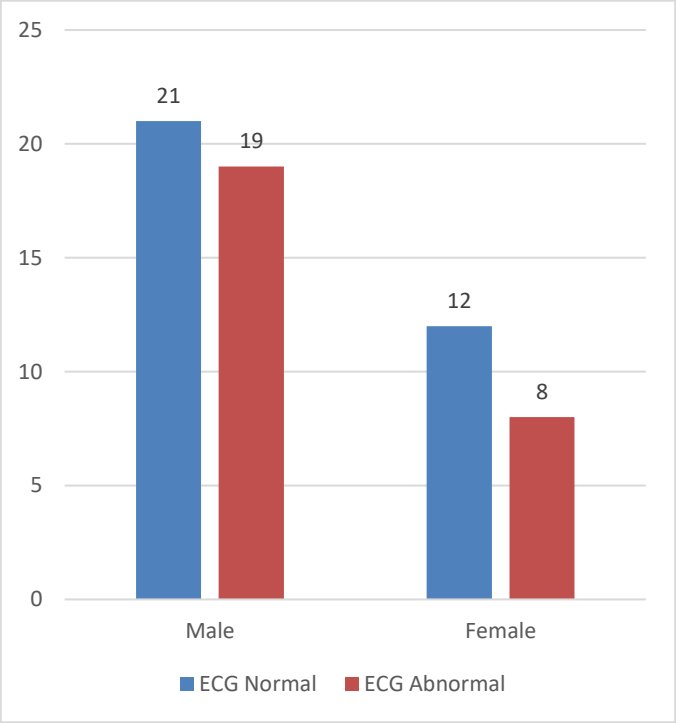
Grade	ECG		Grand Total
	Normal	**Abnormal**	
Grade I	26	2	28
Grade III	2	22	24
Grade II	5	3	8
Grand Total	**33**	**27**	**60**
p<0.0001			



When comparing grade of sting with ECG changes abnormal ECG was found in grade III sting and statistically significant value was obtained and p value was found to be 0.00001.

Table 19 and figure 19:gender with ECG

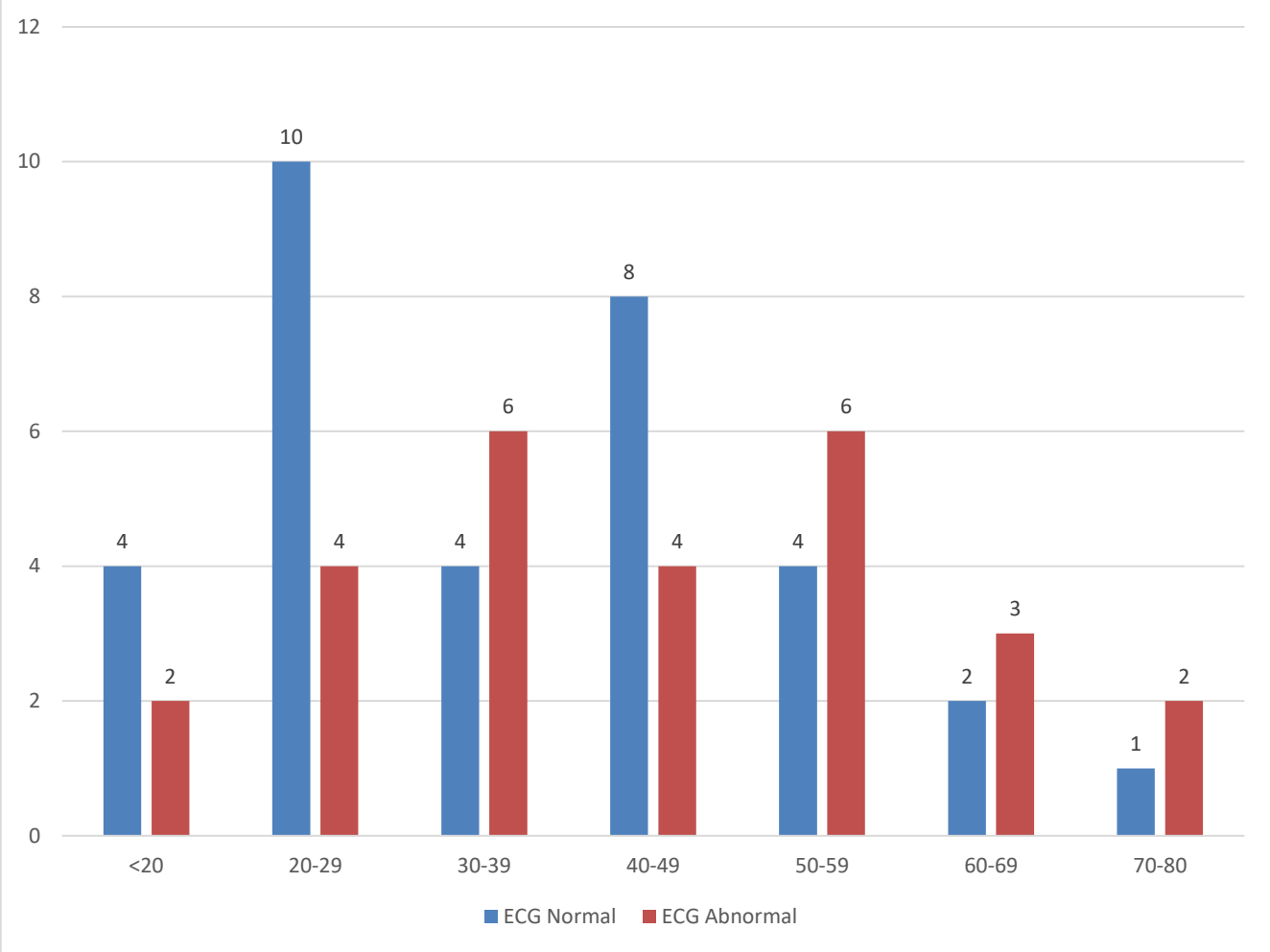
Gender	ECG		Grand Total
	Normal	**Abnormal**	
Male	21	19	40
Female	12	8	20
Grand Total	**33**	**27**	**60**
p=0.58			



When comparing gender with ECG changes p value was found to be 0.58 and it is statistically insignificant.

Table 20 and figure 20:age group vs ECG

Age Group	ECG		**Grand Total**
	Normal	**Abnormal**	
<20	4	2	6
20-29	10	4	14
30-39	4	6	10
40-49	8	4	12
50-59	4	6	10
60-69	2	3	5
70-80	1	2	3
Grand Total	**33**	**27**	**60**
p=0.49			



When comparing age group with ECG p value was found to be 0.49 and it is statistically insignificant and no correlation was found between age group and ECG changes.

DISCUSSION

60 cases were studied in our research, In our study male patients constitute about 66% of cases and female cases contribute to about 33% of cases and it is similar to the study conducted by Abi et al where male cases contribute to about 49% of cases and female cases contribute to about 51% of cases.mild differences may be attributed to the sample size because,sample size in abi et al study is 43.(65)

Out of 60 cases in our study 13 cases had abnormal ECG and 30 cases had normal ECG and it is similar to the study conducted by abi et al ,where out of 43 cases,13 cases had abnormal ECG and 30 cases had normal ECG.

In our study by comparing severity with the ECG changes, statistical test was done and and p value was found to be 0.00001 ,which is statistically significant.(65)

And in our study following ECG changes are noted, Regarding distribution of cases according to ECG changes45% of cases show ecg

changes among that.sinus tachycardia seen in 11 cases,sinus tachycardia with ST depression seen in 4 cases.ST elevation seen in 2 cases,ST depression seen in 2 cases,ST depression with T wave inversion seen 1 case,right bundle branch block seen in 1 case,Tall T waves seen in 3 cases,complete heart block seen in 1 case and left bundle fascicular block seen in 2 cases,

And in the study conducted by abi et al following changes are noted, premature ventricular contractions are seen in 13.9 % of cases, ST depression was noted in 9.3 % of cases,T inversion in 4.6 % of cases ,atrial fibrillation in 4.6% of cases ,U wave in 12.3 % of cases,sinus tachycardia in 11.6 % of cases.(65)

By comparing our study to the study conducted by abi et al,in our study most common ECG change is sinus tachycardia while in reference study is premature ventricular contraction.

In our study statistical significant correlation established between severity with CPK MB level(p value 0.00001),grading with SGOT(p value 0.00001),severity with time (p value 0.02),Time with ECG p values

is 0.003,SGOT with ECG p value is 0.00004,CPK MB with ECG 0.00001, and grading with ECG p value less than 0.000001.

Since significant p values are obtained hence ECG changes and cardiac bio markers can be used as important tool in evaluating ECG changes.

CONCLUSION

In our study 60 cases were studied, and most common age group is 20-29 years of age, male patients accounts for about 66.67% of cases and female patients accounts for about 33.37% of cases.Most common symptom is pain 37 cases had pain and most common sign is tachycardia 22 cases had encountered.

Regarding severity grade 1 in 46% of cases,grade 2 in 13% of cases,grade 3 in 24% of cases.Most common ECG change is sinus tachycardia seen in 18.3% of cases

RBS is elevated in 8% of cases,60% of cases reached hospital in 1-3 hours.CPK MB elevated in 50% of cases,SGOT elevated in 56% of cases

In our study statistical significant correlation established between severity with CPK MB level(p value 0.00001),grading with SGOT(p value 0.00001),severity with time (p value 0.02),Time with ECG p values is 0.003,SGOT with ECG p value is 0.00004,CPK MB with ECG 0.00001, and grading with ECG p value less than 0.000001.

Since significant p values are obtained hence ECG changes and cardiac bio markers can be used as important tool in evaluating ECG changes.

When establishing statistical relationship between RBS with ECG, gender with ECG and age with ECG,p values are found to be 0.24,0.58 and 0.49 and it is statistically insignificant.

Hence since statistical significance established in the cardiac biomarkers and ECG changes,It can be used as marker in evaluation of scorpion sting.

BIBLIOGRAPHY

1.Abroug F, Souheil E, Ouanes I, Dachraoui F, Fekih-Hassen M, Ouanes Besbes L. Scorpion-related cardiomyopathy: Clinical characteristics, pathophysiology, and treatment. Clin Toxicol (Phila). 2015 Jul;53(6):511-8. doi: 10.3109/15563650.2015.1030676. Epub 2015 Apr 8. PMID: 25851549.

2.Yelme G, Jindal A. Scorpion bite, a sting to the heart! Indian J Crit Care Med. 2016 May;20(5):309-10. doi: 10.4103/0972-5229.182198. PMID: 27275084; PMCID: PMC4876657.

3.Alan S, Ulgen MS, Soker M, Geyik F, Karabulut A, Toprak N. Electrocardiologic and echocardiographic features of patients exposed to scorpion bite. Angiology. 2004 Jan-Feb;55(1):79-84. doi: 10.1177/000331970405500111. PMID: 14759093.

4.Elatrous S, Ouanes-Besbes L, Ben Sik-Ali H, Hamouda Z, BenAbdallah S, Tilouche N, Jalloul F, Fkih-Hassen M, Dachraoui F, Ouanes I, Abroug F. Study of severe scorpion envenoming following subcutaneous venom injection into dogs: Hemodynamic and concentration/effect analysis. Toxicon. 2015 Sep 15;104:1-6. doi: 10.1016/j.toxicon.2015.07.003. Epub 2015 Jul 10. PMID: 26166304.

5. Chippaux JP, Goyffon M. Epidemiology of scorpionism: a global appraisal. Acta Trop 2008; 107:71-79.

6. Isbister GK, Bawaskar HS. Scorpion envenomation. N Engl J Med 2014; 371:457-463.

7. Bucaretchi F, Fernandes LCR, Fernandes CB, Branco MM, Prado CM, Vieira RJ, et al. Clinical consequences of Tityus bahiensis and Tityus serrulatus scorpion stings in the region of Campinas, southeastern Brazil. Toxicon 2014; 89:17-25.

8. Freire-Maia L, Campos JA. Pathophysiology and treatment of scorpion poisoning. In: Ownby CL, Odell GV, editors. Natural Toxins, Characterization, Pharmacology and Therapeutics. Proceedings of the 9th World Congress on Animal, Plant and Microbial Toxins. Still water, Oklahoma. Oxford: Pergamon Press; 1989. p. 139-159.

9. Ministério da Saúde. Secretaria Vigilância em Saúde. Departamento de Vigilância Epidemiólogica. Manual de Controle de Escorpiões. Brasília, DF: Ministério da Saúde; 2009.

10. Reckziegel GC, Pinto-Júnior VL. Scorpionism in Brazil in the years 2000 to 2012. J Venom Anim Toxins Incl Trop Dis 2014; 20:46.

11. Gwee MCE, Nirthanan S, Khoo HE, Gopalakrisshnakone P, Kini RM, Cheah L. Autonomic effects of some scorpion venoms and toxins. Clin Exp Pharmacol Physiol 2002; 29:795-781.

12. Meki AR, Mohey El-Dean ZM. Serum interleukin-1β, interleukin-6, nitric oxide and α-1 antitrypsin in scorpion envenomed children. Toxicon 1998; 36:1851-1859.

13. Magalhães MM, Pereira ME, Amaral CF, Rezende NA, Campolina D, Bucaretchi F, et al. Serum levels of cytokines in patients envenomed by Tityus serrulatus scorpion sting. Toxicon 1999; 37:1155-1164.

14. Fukuhara YDM, Reis ML, Dellalibera-Joviliano R, Cunha FQC, Donadi EA. Increased plasma levels of IL-1β, IL-6, IL-8, IL-10 and TNF-α in patients moderately or severely envenomed by Tityus serrulatus scorpion sting. Toxicon 2003; 41:49-55.

15. De-Matos IM, Talvani A, Rocha OO, Freire-Maia L, Teixeira MM. Evidence for a role of mast cells in the lung edema induced by Tityus serrulatus in rats. Toxicon 2001; 39:863-867.

16. Abroug F, Nouira S, El Atrous S, Besbes L, Boukef R, Boussarsar M, et al. A canine study of immunotherapy in scorpion envenomation. Intensive Care Med 2003; 29:2266-2276.

17. Nouira S, Elatrous S, Besbes L, Boukef R, Devaux C, Aubrey N, et al. Neurohormonal activation in severe scorpion envenomation: correlation with hemodynamics and circulating toxin. Toxicol Appl Pharmacol 2005; 208:111-116.

18. Ministério da Saúde. Fundação Nacional da Saúde. Manual de Diagnóstico e Tratamento de Acidentes por Animais Peçonhentos. Brasília: Ministério da Saúde; 1998. 131p.

19. Cupo P, Azevedo-Marques MM, Hering SE. Escorpionismo. In: Cardoso JLC, França FOS, Málaque CMS, Haddad Jr V, editors. Animais Peçonhentos no Brasil – Biologia, Clínica e Terapêutica dos Acidentes. São Paulo: Servier; 2003. p. 198-210.

20. França FOS, Medeiros CR, Málaque CMS, Duarte MR, ChudzinskiTavassi AM, Zannin M et al. Acidentes por Animais Peçonhentos. In: Martins MA, Carrilho FJ, Alves VAF, Castilho EA, Cerri GG, Wen CL, editors. Clínica Médica, Volume 7:

Alergia e Imunologia Clínica, Doenças de Pele, Doenças Infecciosas. Barueri, SP: Manole; 2009. p. 553-613.

21. Mazzei-de-Dàvila CA, Dàvila DF, Donis JH, de-Bellabarba GA, Vilarreal V, Barboza JS. Sympathetic nervous system activation, antivenin administration and cardiovascular manifestations of scorpion envenomation. Toxicon 2002; 40:1339-1346.

22. Pardal PP, Castro LC, Jennings E, Pardal JS, Monteiro MR. Epidemiological and clinical aspects of scorpion envenomation in the region of Santarem, Pará, Brazil. Rev Soc Bras Med Trop 2003; 36:349-353.

23.Bawaskar HS, Bawaskar PH. Vasodilators: scorpion envenoming and the heart (an Indian experience). Toxicon. 1994 Sep;32(9):1031-40. doi: 10.1016/0041-0101(94)90386-7. PMID: 7801338.

24.https://owlcation.com/stem/The-Most-Dangerous-Scorpions-in-the-World

25. Pardal PP, Ishikawa EA, Vieira JL, Coelho JS, Dorea RC, Abati PA. Clinical aspects of envenomation caused by Tityus obscurus (Gervais, 1843) in two distinct regions of Para state, Brazilian Amazon basin: a prospective case series. J Venom Anim Toxins Incl Trop Dis 2014; 20:3.

26. Torrez PP, Quiroga MM, Abati PA, Mascheretti M, Costa WS, Campos LP, et al. Acute cerebellar dysfunction with neuromuscular manifestations after scorpionism presumably caused by Tityus obscurusin Santarem, Para/Brazil. Toxicon 2015; 96:68-73.

27. Boyer LV, Theodorou AA, Berg RA, Mallie J, Chavez-Mendez A, García-Ubbelohde W, et al. Antivenom for critically ill children with neurotoxicity from scorpion stings. N Engl J Med 2009; 360:2090-2098.

28. Bergman NJ. Clinical description of Parabuthustrans vaalicus scorpionism in Zimbabwe. Toxicon 1997; 35:759-771.

29. Campos JA, Costa DM, Franco MM, Oliveira JS. Neurologic manifestations in poisoning by scorpion's bite in infancy. Rev Assoc Med Minas Gerais 1982; 33:8-10.

30. Fernandez-Bouzas A, Morales-Reséndiz ML, Llamas-Ibarra F, Martínez-López M, Ballesteros-Maresma A. Brain infarcts due to scorpion stings in children: MRI. Neuroradiology 2000; 42:118- 120.

31. Gueron M, Stern J, Cohen W. Severe myocardial damage and heart failure in scorpion sting. Report of fi ve cases. Amer J Cardiol 1967; 19:719-726.

32. Gueron M, Yaron R. Cardiovascular manifestations of severe scorpion sting. Clinicopathological correlations. Chest 1970; 57:156-160.

33. Cupo P, Jurca M, Azevedo-Marques MM, Oliveira JS, Hering SE. Severe scorpion envenomation in Brazil. Clinical, laboratory and anatomopathological aspects. Rev Inst Med Trop Sao Paulo 1994; 36:67-76.

34. Gueron M, Ovsyshcher I. What is the treatment for the cardiovascular manifestations of scorpion envenomation? Toxicon 1987; 25:121-130.

35. Freire-Maia L, Campos JA. Response to the letter to the editor by Gueron and Ovsyshcher on the treatment of the cardiovascular manifestations of scorpion envenomation. Toxicon 1987; 23: 123-130.

36. Rona G. Catecholamine cardiotoxicity. J Mol Cell Cardiol 1985; 17:291-306.

37. Simons M, Dowing SE. Coronary vasoconstriction and catecholamine cardiomyopathy. Am Heart J 1985; 109:297-304.

38. Teixeira AL, Fontoura BF, Freire-Maia L, Machado CR, Camargos ER, Teixeira MM. Evidence for a direct action of Tityus serrulatus scorpion venom on the cardiac muscle. Toxicon 2001; 39:703-709.

39. Gueron M, Margulis G, Sofer S. Echocardiographic and radionuclide angiographic observations following scorpion envenomation by Leiurus quinquestriatus. Toxicon 1990; 28: 1005-1009.

40. Amaral CF, Lopes JA, Magalhães RA, de-Rezende NA. Electrocardiographic, enzymatic and echocardiographic evidence of myocardial damage after Tityus serrulatus scorpion poisoning. Am J Cardiol 1991; 67:655-657.

41. Hering SE, Jurca M, Vichi FL, Azevedo-Marques MM, Cupo P. “Reversible cardiomyopathy” in patients with severe scorpion envenoming by Tityus serulatus: evolution of enzymatic, electrocardiographic and echocardiographic alterations. Ann Trop Paediatr 1993; 13:173-182.

42. Abroug F, Boujdaria R, Belghith M, Nouira S, Bouchoucha S. Cardiac dysfunction and pulmonary edema following scorpion envenomation. Chest 1991; 100:1057-1059.

43.https://www.scielo.br/j/rsbmt/a/C6vMnTMvwrwXmBSWjySsVYm/?lang=en&format=pdf

44 https://imagebank.hematology.org/image/63061/urine-findings-in-dic-scorpion-sting

45. Bawaskar HS, Bawaskar PH. Sting by red scorpion (*Buthus tamulus*) in Maharashtra State, India: A clinical study. Trans Roy Soc Med Hyg 1989; 83: 858-860.

46.Rajarajeswari G, Sivaprakasam S, Viswanathan J. Morbidity and mortality pattern in scorpion sting–a review of 68 cases. J Indian Med Assoc 1979; 73: 123-126.

47. Mahadevan S, Choudhury P, Puri RK, Srinivasan S. Scorpion envenomation and the role of lytic cocktail in its management. Indian J Pediatr 1981; 48: 757-761.

48. Biswal N, Charan MV, Betsy M, Nalini P, Srinivasan S, Mahadevan S. Management of scorpion envenomation. Pediatrics Today 1999; 2: 420-426.

49.Ismail M. The scorpion envenoming syndrome. Toxicon 1995; 33: 825-828.

50. Muller GJ. Scorpionism in South Africa. A report of 42 serious envenomations. South Afr Med J 1993; 83: 405-411.

51.Erfati P. Epidemiology, symptomatology and treatment of buthinae stings. *In*: Arthopod Venoms. Handbook of Experimental Pharmaco-logy. Ed. Bettini S. New York, Springer-Verlag, 1978; pp 312-315.

52.Bawaskar HS, Bawaskar PH. Indian red scorpion envenoming. Indian J Pediatr 1998; 65: 383-391.

53. Bawaskar HS, Bawaskar PH. Prazosin in the management of cardiovascular manifestations of scorpion sting. Lancet 1986; 1: 510-511.

54. Amarai CF, de Rezende NA, Freire-Maia L. Acute pulmonary edema after *Tityus serrulatus* scorpion sting in children. Am J Cardiol 1993; 71: 242-245.

55. Abrough F, Ayari M, Nouira S. Assessment of left ventricular function in severe scorpion envenomation–combined hemodynamic and ECHO-doppler study. Inten Care Med 1995; 21: 629-635.

56.Jamanthal JH, Srinivas HV. Hemiplegia following scorpion sting. Indian Pediatr 1973; 10: 337-339.

57. Bisaria BN, Vasavada JB, Bhatt A, Nair PNR, Sharma VK. Hemiplegia and myocarditis following scorpion bite. Indian Heart J 1977; 29: 97-100.

58.Tiwari SK, Gupta GB, Gupta SR, Mishra SN, Pradhan PK. Fatal stroke following scorpion bite. J Assoc Phys India 1988; 36: 225-226.

59. Naik M, Shukla RC, Varma DN, Gupta SK. Intracerebral hemorrhage following scorpion bite. Neurology 1990; 40: 1801.

60.Nagorka AR, Bergeson PS. Infant methamphet-amine toxicity posing as scorpion envenomtion. Pediatr Emerg care 1998; 14: 350-351.

61.Sofer S, Shalev H, Weizman Z, Shaha KE, Gueron M. Acute pancreatitis in children following envenomation by the yellow scorpion *Leiurus quinquestriatus*. Toxicon 1991; 29: 125-128.

62.Bawaskar HS, Bawaskar PH. Severe envenoming by Indian red scorpion *M. tamulus*–the use of Prazosin therapy. Quart J Med 1996; 89: 701-704.

63.Senapati MK. Treatment of scorpion sting. Brit Med J 1962; 2: 1546.

64. Cutting WAM. Treatment of scorpion sting. Brit Med J 1963; 1: 47

65.Abdi A, Farshidi H, Rahimi S, Amini A, Tasnim Eftekhari SF. Electrocardiologic and echocardiographic findings in patients with scorpion sting. Iran Red Crescent Med J. 2013 May;15(5):446-7. doi: 10.5812/ircmj.2853. Epub 2013 May 5. PMID: 24349739; PMCID: PMC3838661.

PROFORMA

Name : Date /Time:

Age /Sex : I.P.No.:

Circumstances of Exposure :

Time of Exposure ::

Time interval

Location : Urban/Rural Home/Office:

C/F :

Site of bite:

Pain/numbness:

giddiness/breathlessness

O/E :

PR

BP

RR

CVS

RS

CNS

Signs of Peripheral Circulatory Failure

Past h/o : DM/HT/IHD/CKD

Investigation :

TC

DC

RBS :

Urine analysis:

Urea : Creatinine :

ECG:

ECHO:

CR Enzymes: CPK MB

SGOT:

Specialist opinion :

Treatment :

Condition at discharge :

CONSENT FORM

Format for Informed Consent Form for Parent / Guardian of the Subjects

Informed Consent form to participate in a research study

Study Title:

Study Number: _____

Subject's Initials: _____ Subject's Name: _____

Date of Birth / Age: _____

(i) I confirm that I have read and understood the information sheet dated _____ for the above study and have had the opportunity to ask questions. [ ]

(ii) I understand that my son / daughter's participation in the study is voluntary and that he/she is free to withdraw at any time, without giving any reason, without his/her medical care or legal rights being affected. [ ]

(iii) I understand that the Ethics Committee and the regulatory authorities will not need my permission to look at my son / daughter's health records both in respect of the current study and any further research that may be conducted in relation to it, even if he/she withdraws from the trial. I agree to this access. However, I understand that my son / daughter identity will not be revealed in any information released to third parties or published. [ ]

(iv) I agree not to restrict the use of any data or results that arise from this study provided such a use is only for scientific purpose(s). [ ]

(v) I agree for the participation of my son/daughter in the above study. [ ]

Signature (or Thumb impression) of the Subject's parent /Legally Acceptable Guardian
Date: _____/_____/_____
Signatory's Name: _____
Signature: _____
Or
Representative: _____
Date: _____/_____/_____
Signatory's Name: _____

Signature or thumb impression of the Witness: _____
Date: _____/_____/_____
Name & Address of the Witness: _____

நோயாளிகளுக்கு அறிவிப்பு மற்றும் ஒப்புதல் படிவம்
(மருத்துவ ஆய்வில் பங்கேற்பதற்கு)

ஆய்வு செய்யப்படும் தலைப்பு:
பங்கு பெறுவரின் பெயர்:
பங்கு பெறுவரின் வயது:

		பங்கு பெறுவர் இதனை ✓ குறிக்கவும்
1.	நான் மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்களை படித்து புரிந்து கொண்டேன். என்னுடைய சந்தேகங்களை கேட்கவும், அதற்கான தகுந்த விளக்கங்களை பெறவும் வாய்ப்பளிக்கப்பட்டுள்ளது என அறிந்து கொண்டேன்.	☐
2.	நான் இவ்வாய்வில் தன்னிச்சையாக தான் பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த கட்டத்திலும், எந்த சட்ட சிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகி கொள்ளலாம் என்றும் அறிந்து கொண்டேன்.	☐
3.	இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு மேற்காள்ளும் போதும் இந்த ஆய்வில் பங்குபெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். நான் ஆய்வில் இருந்து விலகிக் கொண்டாலும் இது பொருந்தும் என அறிகிறேன்.	☐
4.	இந்த ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக் கொள்ள மறுக்க மாட்டேன்.	☐
5.	இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக் கொள்கிறேன் எனக்கு கொடுக்கப்பட்டஅறிவுரைகளின் படி நடந்து கொள்வதுடன், ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்று உறுதியளிக்கிறேன். என் உடல் நலம் பாதிக்கப்பட்டாலோ, அல்லது எதிர்பாராத, வழக்கத்திற்கு மாறான நோய்குறி தென்பட்டாலோ உடனே இதை மருத்துவ அணியிடம் தெரிவிப்பேன் என உறுதி அளிக்றேன்.	☐

பங்கேற்பவரின் கையொப்பம் / ..... இடம் .....
கட்டைவிரல் ரேகை
பங்கேற்பவரின் பெயர் மற்றும் விலாசம் .....
ஆய்வாளரின் கையொப்பம் /..... இடம் .....
ஆய்வாளரின் பெயர் .....
மையம் .....
கல்வியறிவு இல்லாதவற்கு (கைரேகை வைத்தவர்களுக்கு) இது அவசியம் தேவை
சாட்சியின் கையொப்பம் /..... இடம் .....
பெயர் மற்றும் விலாசம் ....

S.NO	NAME	Age	Sex	IP No	TIME of Admission	Symptoms	Signs	Grade	ECG	ECG (Normal /Abnormal)	RBS	TC	CPK MB	SGOT	Time in Hours interval
1	RAJKUMAR	34	Male	69818	2.40 PM	1,2	Nil	Grade I		Normal	105	8000	16.1	42	1
2	MALATHI	58	Femae	42920	11.00 PM	1,3,6	6	Grade III	2	Abnorma	78	4500	72	120	3
3	SUBBAIAH	80	Male	9919	9.50 PM	1,2	6	Grade I	1	Abnorma	147	11000	15.75	30	0.75
4	KANTHAN	48	Male	10626	6.35 PM	1,6	4	Grade I		Normal	178	9000	16	41	1.5
5	VIGTORIA MARY	43	Femae	16092	5.10 AM	8,10	Nil	Grade I		Normal	107	12000	72	96	2
6	BALAJI	35	Male	17772	4.45 PM	1,3,5	1,5	Grade III	2	Abnorma	88	3800	71.65	38	3
7	BALAN	58	Male	20647	3.55 AM	2,9	6	Grade I		Normal	111	4800	18.5	28	0.5
8	SURYA	17	Male	26382	2.40 PM	4,6,7	1,4	Grade III	1	Abnorma	77	7500	80	25	2
9	SUDALI	36	Femae	14974	6.50 AM	1,2	1,2	Grade III	4	Abnorma	112	12000	15.6	26	1.5
10	SURYA PRAKASH	20	Male	26653	9.30 PM	1,5	6	Grade I		Normal	119	5000	15.75	36	3.5
11	MUNIYA SAMY	27	Male	29111	2.20 PM	1,2	2,6	Grade III	8	Abnorma	75	6000	74.35	95	2.5
12	MADHUMEENA	18	Femae	29372	6.45 AM	8	1,2	Grade II	1	Abnorma	187	8300	15.8	35	1
13	UMA MAHESWARI	25	Femae	35865	1.30 PM	2,9	6	Grade I		Normal	220	6800	16.35	24	6
14	MANIMUTHU LAKSH	32	Femae	36834	3.30 PM	1,3	3	Grade I		Normal	138	12800	16.65	36	1
15	MAHESWARI	23	Femae	47133	4.20 PM	5,7	1,2	Grade III	1	Abnorma	164	13500	74.35	78	3.5
16	SANKARESWARI	45	Femae	50174	4.30 AM	1,2	6	Grade I		Normal	125	15000	16.25	39	1
17	PITCHAIKONAR	72	Male	44812	7.25 PM	1,2	6	Grade I		Normal	85	7300	16	36	2
18	SUDALAI KANNU	54	Male	46803	9.20 PM	5,7	1,7	Grade III	7	Abnorma	154	5200	72	120	4
19	VEERA PANDI	37	Male	46823	10.10 PM	2,9	1,2	Grade III	1	Abnorma	96	11000	18	66	2
20	CHELLAPANDIAN	60	Male	46960	6.35 PM	1,3,4	4,1	Grade III	1	Abnorma	112	5600	78	96	3
21	SUBASH	23	Male	53501	6.00 PM	1,2	4	Grade I		Normal	108	4800	17	40	1
22	KARTHIEESWARAN	30	Male	53617	8.20 PM	1,2	4	Grade II		Normal	93	7200	44	88	2
23	THANGAPANDI	27	Male	55748	9:00 PM	5,7	1,2	Grade III		Normal	200	4300	74	122	3.5
24	ARULJOTHI	27	Male	55957	10.55 PM	1,2	2,6	Grade III	9	Abnorma	210	6900	72.5	84	1
25	MADATHI	50	Femae	40139	4.30 PM	1,2	Nil	Grade I		Normal	86	9000	16.8	46	1.5
26	Kejenthiran	47	Male	41609	10:00 AM	1,3	Nil	Grade I		Normal	75	10200	15.3	37	1
27	MUTHU LAKSHMI	55	Femae	43322	12.50 PM	1,3	6	Grade I		Normal	118	4500	16.5	42	1.25
28	MARIAPPAN	38	Male	43989	9:00 AM	6,7	1,2	Grade III	4	Abnorma	95	6200	72	92	2
29	NAMBIRAJAN	22	Male	91840	1.20 AM	2,9	6	Grade II		Normal	102	11400	15.9	56	1
30	KALAIPANDIAN	41	Male	85286	12.10 AM	4,6,7	1,6,7	Grade III	2	Abnorma	146	6500	82.5	172	3
31	RAMALAKSHMI	65	Femae	45042	12.30 AM	1,2	6	Grade I		Normal	89	4800	21	45	0.5
32	MASANAMUTHU	70	Male	47012	8.50 AM	1,3,9	1,5	Grade III	5	Abnorma	78	5100	74.5	99	5
33	MUPPIDATHI	35	Femae	47138	9.30 PM	1,3	3	Grade I		Normal	102	4600	16.8	42	1.5
34	MAHESH KUMAR	18	Male	55965	8.30 PM	3	4	Grade II		Normal	93	7800	31	36	2
35	RAHUL	17	Male	60423	4.30 AM	1,2	2,6	Grade II		Normal	117	12300	30	45	1
36	SIVAPERUMAL	42	Male	73842	6.15 PM	1,5	1,5	Grade III	7	Abnorma	106	5300	71	95	3
37	GOPI	32	Male	75945	1.10 AM	5,7	1,2	Grade III	1	Abnorma	86	4900	75	100	4
38	MAYILAMMAL	40	Femae	48592	9.45 PM	2,9	1,6	Grade I		Normal	201	5200	16	32	1
39	DHANALAKSHMI	46	Femae	49726	2.30 PM	1,3	Nil	Grade I		Normal	95	4800	16	36	2

S.NO	NAME	Age	Sex	IP No	TIME of Admission	Symptoms	Signs	Grade	ECG	ECG (Normal /Abnormal)	RBS	TC	CPK MB	SGOT	Time in Hours interval
40	MARI RAJ	45	Male	52780	6.35 PM	1,3,4	1,4	Grade III	1	Abnorma	112	6100	76	96	3
41	DEVENDRA RAJA	17	Male	91872	1.30 AM	1,2	6	Grade I		Normal	82	5800	15	32	2
42	**SETHURAJ**	55	Male	91883	9.20 PM	1,2	4	Grade II	3	Abnorma	91	7400	41	88	3
43	VENKATRAJ	54	Male	94069	4:00 AM	6,5	1,7	Grade III	2	Abnorma	146	8900	84	176	4
44	RABIN	21	Male	53918	7.05 AM	7	4	Grade I		Normal	214	8200	66	88	4
45	GANAPATHI PANDIA	60	Male	55905	4.20 PM	5,7	2,4,5	Grade III	7	Abnorma	115	5600	72	120	6
46	NAWASH	23	Male	56153	6.45 AM	2,9	1,2	Grade III		Normal	76	4700	16.5	52	1
47	SUGESH	19	Male	58195	10:00 PM	5,8	6	Grade I		Normal	84	6100	72	68	3
48	NAGARAJAN	52	Male	59568	7.30 PM	1,2	1,2	Grade I		Normal	107	4400	18	44	2
49	PETCHIMUTHU	55	Male	61378	8.10 PM	8,10	4	Grade III	1	Abnorma	126	6900	76	89	3.5
50	VINITHA	24	Femae	62668	10.25 PM	1,2	2,6	Grade III	9	Abnorma	90	7200	59	102	3
51	SHANMUGAPRIYA	20	Femae	62549	12.30 AM	1,2	6	Grade I		Normal	88	4800	16	59	1
52	THIRUMANI	53	Femae	64359	9.40 PM	1,3,9	1,5	Grade II	1	Abnorma	158	10200	18	47	1.5
53	RAMAN	27	Male	64584	10:00 PM	1,3	6	Grade II		Normal	78	6600	16.5	38	1
54	JESURAJ	42	Male	69768	4:00 PM	1,2	Nil	Grade I		Normal	94	4200	15.7	42	2
55	SELVIN	37	Male	76935	7.35 PM	1,3	6	Grade I	1	Abnorma	104	9400	64	86	1.75
56	JULIET	47	Femae	75893	7:00 AM	7,5	1,2	Grade III	3	Abnorma	116	11000	74.5	122	3.5
57	VIGNESH WARAN	27	Male	78024	10:00 PM	1,3	6,2	Grade I		Normal	191	6100	74	160	2
58	RAJAMMAL	63	Femae	80760	11.30 PM	6,7	1,2	Grade III	6	Abnorma	79	4500	81	116	2
59	VALLIAMMAL	60	Femae	85501	2.15 AM	1,2	Nil	Grade I		Normal	120	7300	16.2	43	1.5
60	DARMARAJ	49	Male	86279	4.15 PM	1,2	Nil	Grade I		Normal	153	4200	15.8	38	1

KEY SYMPTOMS :-

1 -Local pain, 2 - Local swelling, 3-Paraesthesia, 4-Chest pain, 5-Sweating, 6-Palpitations ,7-Breathlessness, 8-Giddiness, 9-Redness at site 10-vomiting

KEY SIGNS:-

1-Tachycardia, 2-Hypotension, 3-Hypertension, 4-Profuse sweating, 5-Cold peripheries, 6-Tenderness at sting site, 7-Pulmonary edema

ECG:-

1-sinus tachycardia, 2-sinus tachycardia with ST depression, 3-ST elevation, 4-ST depression, 5-ST depression with T wave inversion 6-Right bundle branch block, 7-Tall T waves, 8-Complete heart block, 9-Left anterior fascicular block