Scorpion envenomation is a major public health problem in tropical and sub-tropical countries, especially in Africa, Middle-East, Latin America and India. At times it poses a significant life-threatening acute time limiting cardiovascular emergency. Irrespective of different species of scorpion, similar cardiovascular effects are reported. Irrespective of the understanding of the pathophysiology and management, the morbidity and fatality remains high in rural areas due to non-existent medical facilities and the delay in hospitalisation due to superstitions and faith in village healers. Scorpion envenoming has been under-estimated as this problem is faced by majority of underdeveloped and developing countries. Since the advent of prazosin therapy the fatality has dropped from 29% to less than 1%.

The Indian red scorpion *Mesobuthus tamulus* (Figure 1) is the most lethal amongst all the poisonous species of scorpions. The scorpion envenomation has been frequently reported from Puducherry, Karnataka, Tamil Nadu, Andhra Pradesh, Saurashtra, Uttar Pradesh, Bihar and Marathwada and western Maharashtra. Thousands of scorpion stings are reported annually from India and 15% to 20% of these sting manifests with systemic symptoms. Farmers are more prone to get stung by scorpion during handling debris and paddy husk in the months of April to early June and September to October as due to sudden rise in environmental temperature, scorpions come out of their hides. The sting during these months is more potent and prone to serious envenomation. Scorpion is nocturnal in habit and people walking bare foot become their victim more commonly.

**SCORPION**

Nearly 1,000 species of scorpion are known worldwide, which belongs to six families. However, only the scorpion belonging to the family *Buthidae*, secretes neurotoxic venom that is toxic to human; around 86 species of this family are found in India. *M. tamulus*, an Indian red scorpion, is venomous and its envenomation is fatal if not treated in time. Its claws are red coloured, put tail, legs and body is covered with khaki coloured cuticles. It is 2.5 to 4 inches in length. The tail consists of stout segments with terminal bulb containing pair of telson venom secreting salivary glands. It actively secretes venom at the time of sting by a sharp semi-curved stinger. The stinger is 2 to 4 mm size and human skin thickness is 1.5 to 4 mm. Scorpion venom is rich in neurotoxin.

The Black scorpion *Palmaneus gravimanus* is less poisonous (Figure 2). It is seen in Kerala, Vidharbha and Marathwada region of India. It is bigger in size as compared to red scorpion. It inflict severe excruciating painful sting. Its claws are broad and thick and strong while tail consists of thin segments.
Scorpion Venom

All scorpion species secrete venom. Venom is a mixture of various active substances; of these neurotoxins are the most important. Neurotoxins consist of different small sized proteins with sodium and potassium cations, which interfere with the neurotransmission. Venom action on neurotransmitter is rapid and fast. It contains peptide neurotoxin that opens the sodium channels (beta-toxin). The scorpion venom depolarises the cell membrane; in addition it also inhibits the deactivation of sodium channels (alpha-toxin). There is massive release of endogeneous catecholamines in to the circulation due to delayed activation of sodium neuronal channel by the venom. The venom of the *Mesobuthus tamulus* (Indian red scorpion), *Buthus martensi* (Chinese scorpion) and *Leiurus quinquestriatus* (yellow scorpion) causes autonomic storm by stimulating autonomic nervous system (ANS). Charybdotoxin another component of the venom inhibits the calcium dependent potassium channels; similarly iberiotoxin isolated from *M. tamulus* has similar action on potassium channels. Scorpion venom also contains serotonin, which may cause local pain at the site of the sting. The venom of Tityus species has a kallikrein inhibitor causing raised bradykinin levels. Venom of *Tityus serrulatus* from Trinidad is pancreotoxins, responsible for the development of acute pancreatitis.

**CLINICAL MANIFESTATIONS**

Clinical effects of the envenoming depend upon the species of scorpion and dose of venom injected at the time of sting. The severity of envenoming is related to age, size of scorpion and the season of the sting. High fatality rate is seen in children. The early or pre-monitory clinical manifestations characterised by vomiting, profuse sweating all over the body (Figure 3), priapism (Figure 4), cold extremities and mild tolerable pain. This pain becomes severe excruciating when extremities become warm, a sign of recovery.

Clinical presentations can be divided into following two grades.

1. **Local manifestations:** severe local pain without systemic involvement
2. **Systemic manifestations.**

**Local Manifestations**

Local pain or severe excruciating pain is the only clinical manifestation seen in 35% of cases in a recent series. In 57%, 33% and 11% cases; lower extremities, upper extremities and the other part of the body are the sites of sting, respectively. Severe pain radiates along the corresponding dermatomes. Local oedema, urticaria, fasciculation and spasm of underlying muscles are seen at the site of sting due to persistent stimulation of pain conducting receptors and the liberated serotonin. Due to pain there is transient bradycardia, transient rise in blood pressure and mild sweating with warm extremities. Sudden tap at the site of sting induces severe pain and sudden withdrawal of the part called “tap sign”. Patient continuously moves the stung part of body or holds it firmly to find a comfortable position.

**Systemic Manifestations**

Soon after scorpion sting the victim suffers from the autonomic storm and shows all pre-monitory signs and symptoms. The red scorpion venom is potent sodium channel activator and stimulates the ANS resulting in sudden pouring of catecholamines in the circulation. Both sympathetic and parasympathetic twigs are stimulated leading to “autonomic storm”, characterised by initial parasympathetic stimulation clinically detected in form of vomiting once or twice, profuse sweating all over the body (4 to 12 hours), ropy salivation (Figure 5), priapism (6 to 8 hours), mydriasis, bradycardia, hypotension, transient premature ventricular beats. Sympathetic stimulation is characterised by propped eyes, puffy and anxious facies, oculogyric crisis, chest discomfort, perioral paraesthesias, at times tingling and numbness all over body and cool extremities.

![Figure 3: Profuse sweating.](image)

![Figure 4: Priapism.](image)

![Figure 5: Ropy salivation.](image)
Skin over hands, feet, palm and sole look like a washer-man hands as they appears finely wrinkled and cold. Clinical manifestations at times are apparently diverse irrespective of similar pathology. In some patients there is minimal local pain on arrival but as the peripheral circulation improves with time and warming, they start experiencing excruciating pain. Severe transient muscle spasm may occur due to the loss of electrolytes. Clinical manifestations depends upon the time elapsed between sting and hospitalisation and the treatment received at periphery. The major manifestation includes hypertensive crisis and pulmonary oedema (PE) which may be fatal if not treated timely.

**Hypertension**

Upto 45% of victims with systemic involvement were found to have hypertension when reported within 15 minutes to 11 hours after the sting. Children may present with agitated look, confusion, generalised convulsion, transient hemiplegia, oculogyric crisis, bilateral extensor planter response, propped up eyes and a puffy face suggestive of hypertensive crisis. It may be difficult to measure accurate blood pressure in such children. Majority of these cases complained of headache, chest discomfort, suffocation, giddiness and paraesthesia.

Transit initial hypotension is due to hypovolaemia secondary to acetylcholine excess, it is further aggravated by hot climatic condition in summer months of the tropics.

**Pulmonary Oedema**

PE occurs in upto 30% severe scorpion sting cases with respiratory failure. PE develops within 30 minutes to maximum 36 hours. In upto 8% cases massive PE may be life-threatening. Clinically PE can be suspected when respiratory rate is >24 per minute, orthopnoea, intractable cough, low volume fast thready pulse, narrow pulse pressure, summation gallops, systolic murmur and moist basal rales, central cyanosis. Intractable cough with massive expectoration of blood mixed froth from the mouth and nostril with central cyanosis, hypo- or hypertensive and loud death rattle sound heard are suggestive of massive PE.

Alpha receptors stimulation plays an important role in the pathogenesis of PE. Disseminated intravascular coagulation, coma, convulsion, miosis, mydriasis and hemiplegia, aphasia, cerebral infarction, transient bilateral cerebellar syndrome and subdural hematoma have been reported during pre-prazosin era. Acute renal failure due to scorpion sting is rare. However, ill-treated, delayed reporting of a case may result in death due to multi-system organ failure and acute respiratory distress syndrome.

After 20 to 30 hours of recovery from autonomic storm, the victim develops asymptomatic warm extremities, accompanied by bradycardia, hypotension with prolonged QTc interval (0.50-0.65 msec) on electrocardiogram (ECG). He may look exhausted. The depletion of tissue catecholamines following autonomic storm is the cause and patient usually recovers within 72 to 96 hours without any intervention. This phenomenon does not occur if victim receives scorpion antivenom and prazosin simultaneously.

**Fatality**

Victim can die suddenly due to ventricular arrhythmias which usually occur within 15 to 30 minutes of sting. Many victim die from severe PE, brain haemorrhage and multi-system organ failure, if not treated properly and in time.

**INVESTIGATIONS**

Total leukocyte counts are raised to 11,000-26,000 due to venom induced interleukin-6 and tumour necrosis factor-α release. Cardiac CPK MB is raised. Reduction in serum amylase and serum calcium levels may be observed. There is raised serum glucose, potassium and reduction in insulin level.

Chest radiograph may reveal unilateral or bilateral batwing or patchy PE (Figure 6). At times secondary respiratory infection in the form of consolidation is seen in a hospitalised patient recovering from PE. Mild cardiomegaly may be present. In author’s series of cases of PE due to scorpion sting, the follow-up of more than 15 years did not show any cardiovascular abnormality.

**Electrocardiogram**

ECG is one of the most important diagnostic and easily available investigation. Not a single victim with systemic involvement has normal ECG. Sinus bradycardia seen in early hypertensive cases with a heart rate of 42 to 60 per minute, usually persisted for 3 to 4 hours. The other common findings are ventricular premature contraction, couplets, transient non sustained ventricular tachycardia and rarely fatal ventricular arrhythmias (Figure 7). The sinus tachycardia, injury to conducting system in the form of left anterior hemiblock, bundle branch block, complete heart block and marked tented T waves may be the other common findings. The tall T waves may mimic acute myocardial infarction. Elevated ST segment with small q wave is also often seen in lead I and AVL. Severe PE cases showed low voltage pattern with PQRS T alterans with ST segment depression. Recovering victim's ECG showed large T wave with wide base and round top suggestive of delayed depolarisation with prolonged QTc, accompanied with asymptomatic bradycardia and hypotension. The severity of ECG changes did not correlate with clinical condition.

**Echocardiography**

Echocardiography shows poor global contractility 12-15 hours after sting with low ejection fraction, decreased systolic ventricular performance and mitral incompetence. Abnormal diastolic filling persisted for five days to four weeks. There is good echocardiographic correlation between clinical improvement and return of left ventricular function.
Mild envenomation causes severe vasoconstriction and hypertension; while predominant left ventricular dysfunction with normal systemic vascular resistance with PE is seen in severe scorpion sting. However, severe hypotension depends upon the fluid balance, while hypotension and shock with warm extremities occurs in terminal stage due to biventricular dysfunction and terminal vasodilation (warm shock).

**MANAGEMENT**

No scorpion sting should be taken as benign unless observed for 24 hours, irrespective of species involved. On the basis of pathophysiology, therapeutic effort should be directed against venom, the clinical manifestations of the over stimulated ANS and after effects of excessive catecholamine and correction of hypovolaemia. The incision at the sting site or tourniquet is not advisable and patients who come for medical care after 4 hours of sting and do not show signs of systemic envenomation can be treated symptomatically without prazosin or scorpion antivenom.

**Local Envenomation**

Mild pain can be abolished by application of ice packs over the site of sting. Severe excruciating pain can be transiently relieved by local anaesthesia (lignocaine without adrenaline). However, oral diazepam and non-steroidal anti-inflammatory drugs (NSAIDs) with first initial dose of lignocaine can give prolonged relief from pain. At times severe intolerable pain, patient tossing in bed without signs of autonomic storm can be given injectable opioid (pentazocine).

**Systemic Envenomation**

Dehydration leading to hypovolaemia is due to vomiting, excessive salivation and profuse sweating. It should be corrected by oral rehydration. Intravenous crystalloids or hydration by nasogastric tube may be necessary in a confused and agitated victim. Fluid deficit must be corrected since hypovolaemia is one of the proposed mechanisms of shock syndrome in scorpion sting. Electrolytes imbalance should also be corrected.

**Prazosin:** An $\alpha-1$ adrenergic receptor blocker reduces preload, left ventricular impedance without causing tachycardia. It reverses the metabolic syndrome evoked due to excessive catecholamines release. Prazosin is a pharmacological and physiological antidote to scorpion venom actions. It also inhibits sympathetic outflow in CNS. It inhibits phosphodiesterase, thereby enhancing a cGMP level which is one of the mediators of nitric oxide synthesis. It enhances insulin secretion which is inhibited by venom. Thus, its pharmacological properties can antagonise the haemodynamic, hormonal and metabolic effects of scorpion venom (Figure 8). It can be administered orally in a dose of 250-500 µg/Kg in children and 500-1000 µg/Kg in adults and should be repeated every three hourly until the signs of clinical improvement appear or till dry and cool extremities persist. If the initial dose has been vomited, it should be repeated. Due care should be taken to avoid postural fall in blood pressure.
which is a known side-effect of prazosin (first dose phenomenon). Postural hypotension should be treated by lowered head position and intravenous fluids. Since the advent of prazosin the fatality due to severe scorpion sting has reduced to less than 1%. Prazosin is poor man scorpion antivenom.

**Scorpion antivenom**

It is available for clinical use. Venom causes transient parasympathetic and prolonged sympathetic stimulation. Ongoing cholinergic phenomenon is suggestive of free circulating scorpion venom, which can be neutralised by antivenom, while sympathetic stimulation suggests after effects and fatality is due to sympathetic over activity. Scorpion antivenom is effective if a victim is brought in a stage of acetylcholine excess that is within 1 to 4 hours of sting. Recovery time is shortened in a case treated with scorpion antivenom and prazosin than prazosin alone. Total dose of antivenom required is 30 to 100 mL. However, scorpion antivenom is expensive and always in short supply. No test dose is required as there are high circulating catecholamines and anaphylaxis is very rare.

PE is the most important cause of mortality and should be treated with propped up position, nasal oxygen, intravenous loop diuretics, oral prazosin. Massive PE may also require immediate oral nitroglycerine (NTG) spray to reduce the pulmonary congestion and intravenous NTG drip or if available sodium nitroprusside 4-5 µg/kg/minute. Blood pressure should be closely monitored and maintained at the level of systolic 80-90 mmHg. Inotropic support with dopamine and dobutamine 5-15 mg/kg/minute is advocated for 36 to 48 hours in warm hypotensive shock patients. Unconscious patients with cardiorespiratory failure may also require invasive or non-invasive mechanical ventilatory support. A stepwise management of scorpion sting is shown in Figure 9.

Cardiac arrhythmias are many times self-limiting. Intravenous amiodarone should be used with caution as it may precipitate massive PE, marked bradycardia and cardiac arrest. Captopril, glucose insulin potassium drip and L-carnitine have also been tried to alleviate the venom effects on the heart but does not approved indication.

Atropine, steroids, antihistamines, beta blockers, calcium channel blockers, excessive diuretics, adrenalinics and narcotics are to be avoided, as. These do more harm than good in scorpion envenoming.

**PREVENTION**

Scorpions are killed by organophosphorus pesticides. A false ceiling of plastic sheet should be put under the roof of hut to prevent scorpions falling in bed from loose tiles of roof. Shoes and clothes should be checked before wearing. Shoes should be packed with paper or cloth so as to prevent the entry of scorpion in night. Hand gloves made of thick rubber should be worn while harvesting fire wood, dry cow dung, lifting paddy, Jawar (sorghum) and sugar cane husk. Children should not be allowed to go outside in early darkness without shoes. Simple sandal or slipper did not prevent the sting. One should not put hand blindly in crevices, doors or old storage material during night hours and bedding or cot should kept at distance from mud house wall and one should not backrest on mud wall.

**RECOMMENDED READINGS**
