REVIEW ARTICLE

Scorpion Sting: Current Management

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Summary: Acute time limiting life threatening refractory pulmonary edema even at times fatal is more painful than severe local intolerable pain what layman even including medical personals knew about scorpion envenoming. We are tudying severe scorpion ting cases since 1976. Envenoming by Mesobuthus tamulus (an Indian red scorpion) result in autonomic torm, characterized by vomiting, profuse sweating, cold extremities and life threatening cardiovascular efects. We observed hypertension in 39%, pulmonary edema 29% and severe local pain at the site of ting without sytemic efects in 24% cases. Fatality is 0.9% in adults and 7% in children. Prazosin a potsynaptic alpha-1 blocker antagonizes the venom action and act as physiological and pharmacological antidote. Since the advent of prazosin the fatality is reduced to <1% which was more than >40% during pre-prazosin era (1961-83). Cardiovascular morbidity and mortality depends upon the age of victim, season of ting, content of telson (poisonous glands situated at the terminal segment of tail of scorpion attached with sharp curved tinger) at the time of ting and time lapse between ting and adminitration of prazosin.

Introduction:

Nearly 1000 species of scorpions belonging to six families have been mentioned, but only some species belonging to the family Buthidae, produces neurotoxin venom that is potent lethal toxic to human victims. Of 86 species found in India Mesobuthus tamulus (an Indian red scorpion) and Palmaneus Garvimanus black scorpion (vernacular language called Ingali) often seen all over keral tate are the common ones. Mesobuthus tamulus, is the mot lethal species lourished all over wetern Maharashtra, Anantpur and Karnool ditricts of Andhra Pradesh, Chennai, Pondicherry and Madurai in Tamil Nadu, Bellary in Karnataka, part of Gujarat, Patna area from Bihar. Recently few deaths have been reported from ret of Maharashtra because of change in environmental temperature, routine use of peticides, and chemical manures by farmers, result in change in venom as a part of acclimatization. Farmers, farm laborers are more prone to get tung by scorpion during handling debris, paddy husk in the months of October- September. Bare feet walking young children in early darkness are at high risk of getting tung, simple wearing of slippers do not protect from ting. Scorpions often fall from loose tiles of huts in beds. Scorpion takes shelter in clothes (shirt shelves, trouser pockets), bedding and

Culprits

Palmaneus Garvimanus (Big black scorpion)

It inlicts severe excruciating painful ting, mild swelling, sweating, and local fasciculation at the site of ting, transient bradycardia due to pain. No sytemic involvement.

Mesobuthus tamulus (An Indian red scorpion)

It has red color claws, tails and legs with body cover with khaki color of size 2.5-4 inches long. Similar lethal scorpion species capable of inlicting fatal ting are reported from north Africa, the Middle Eat, south Africa, Brazil, Trinidad, Mexico and Turkey. Despite zoological diferences among various species, the clinical presentation following envenomation is quite identical.

Venom

Scorpion venom contains polypeptides, free amino acids, serotonin, hyaluronidase and various enzymes, which act on trypsinogen. It is the polypeptide, which is neurotoxic. P subtance timulates the cutaneous pain ibers. The mesobuthus tamulus venom is sodium channel activator and causes delay closing of sodium channels resulting in autonomic torm. The toxic content of venom causes inhibition of calcium dependent potassium channels.

Clinical manifetations

Clinical features can be divided in to two groups

1- Severe local pain at the site of ting radiating along corresponding dermatomes without sytemic involvement.

2- Mild local pain or at times absent but present with severe sytemic involvement

Local pain or benign ting

Sting by less poisonous scorpion species like Palmaneus Garvimanus (black scorpion) or scorpion with empty venom glands called telson are characterized by sudden onset of severe tolerable mild or at times absent local pain, but tart getting severe intolerable pain at the site of ting as soon as there is improvement in peripheral circulation, warming of cool extremities, accompanied with clinical improvement. Local sweating, swelling and fasciculation are seen over the part of ting. Due to severe pain victim tries to move the afected part of body (site of ting) jut to ind comfortable position but eventually fails.

dermatomes. Simple taping over the tung part induces severe pain and withdrawal. Local swelling is often increased by repeated local injections. Many times patient apply a tight tourniquet near the ting site or apply some herbal remedies or even burn the part of ting due to intolerable local pain. Because of severe pain patient is anxious, mild sweating or transient raised blood pressure with bradycardia can be noted, but there are no cold extremities or peripheral vasocontriction or signs and symptoms suggetive of autonomic torm.

Sytemic manifetations

The red scorpion venom is a potent sodium channels activator. Sting injects the venom deep in skin and timulates the autonomic nervous sytem resulting in sudden pouring of catecholamines in to circulation. Both sympathetic and parasympathetic twigs are timulated. But body is not at all prepared for such sudden accident as a result it responds randomly. Autonomic torm is characterized by initial parasympathetic timulation clinically detected in form of vomiting once or twice, profuse sweating from all over the body, sweat literally lows for 7-17 hours (skin diarrhea), hyper-salivation for 2-12 hours, priapism for 6-18 hours, mydriasis, bradycardia (42-60 beats per minute), hypotension, transient ventricular premature beats with bigeminy pattern. Sympathetic timulation is characterized by propped eyes, pufy and anxious face, oculo-gyric crisis, chet discomfort, perioral parathesias at times tingling and numbness (parathesia) all over body and cool extremities. Skin over hand and feet, palm and sole look like a washer-man hand i.e. ine wrinkles and cold. These changes occur simultaneously with parasympathetic timulation but persit for long duration. Para-ternal sytolic lift, transient sytolic murmur of mitral regurgitation due to papillary muscle dysfunction due to coronary spasm is seen. Mild cases have little symptoms with cool extremities without severe sweating. The clinical manifetations at times are apparently diverse irrespective of similar pathology. Another clinically intereting inding observed is that on arrival patients with these symptoms and signs have tolerable mild or at times absent local pain, but tart getting severe intolerable pain as soon as there is improvement in peripheral circulation, warming of cool extremities, accompanied with clinical improvement.

Hypertension

Patients can have raised blood pressure up to 210/160 mm hg with sinus bradycardia within 1-4 hours of ting. Children and occasionally adults with hypertension tend to be confused, agitated, at times have generalized convulsions, transient hemiplegia and oculogyric crisis. Bilateral extensor planter response, severe headaches, propped up eyes and pufy face are important diagnotic signs in children suggetive of hypertensive crisis. It is diicult to measure accurate blood pressure with routine sphygmomanometer in confused agitated child. Victim at times develops massive life threatening pulmonary edema with froth in mouth and notril and intractable cough with hypertension.

Pulmonary edema

This is a common cause of fatality in scorpion ting victims, if not intervened in time. It occurs 4-36 hours after ting. Even sudden development of pulmonary edema in a hospitalized patient has been reported necessitating a close vigilance and monitoring of these victims. Clinically pulmonary edema can be suspected when respiratory rate is >24 per minute, orthopnea, intractable cough, low volume fat thready pulse, summation gallops, sytolic murmur and basal moit rales which may be auscultated over the dependent part or at times all over chet. 12% victims remain untreated or report too late after ting or at periphery if they received atropine, excessive teroids, antihitamine, digoxin and intravenous luid or massive doses of furosemide develop acute dyspnea, cyanosis, bring copious massive blood tained froth in mouth and notril. Moit bubbling rales with tracheal death rattles are heard all over chet. At times this patient has marked tachycardia with hypotension.

Tachycardia

In hospitalized children recovering from vasocon-triction usually after 12-18 hours develop marked tachycardia (heart rare 110-240 per minute) with warm extremities. Such child has air hunger, delir-ium, may throw convulsive movement, moit basal rales in chet, summation gallops with murmur in the heart. After 20-30 hours in a case recovering from autonomic torm, he develops warm extremi-ties, bradycardia (heart rate 50-60 per minute), split second heart sound with hypotension (70-90 mm Hg) with prolonged QTC (500-650 msec.) with no complaints. This is result of depletion of tissue cate-cholamines due to autonomic torm, usually it takes 72-96 hours for recovery.

Other features are pain in abdomen which may be due irritation of worm (round worm infetation is common in villagers) by scorpion venom, in addition to smooth muscle contraction due to acetylcholine excess. Hyperglycemia, raised serum amylase

suggetive of acute pancreatitis is transient and reversible. Hemiplegia, aphasia, cerebral infarction and subdural hematoma due to disseminated intra-vascular coagulation have been reported. Acute renal failure has also been reported. Persitent or prolonged hypoxia due to low output tate, marked tachycardia, respiratory failure due to pulmonary secretion and edema may result in irreversible cerebral anoxic injury with subsequent persitent cerebral insuiciency and low intelligence ret of life.

Fatality
Victim can die suddenly due to lethal ventricular arrhythmias, which occur within 15-30 minutes of ting. Many cases died due persitent pulmonary edema, tachycardia, hypotension and convulsion and brought to hospital in moribund, comatose condition, pin point pupils with massive pulmonary edema.

Invetigations
Total leucocytes count is raised to 14000-21000/ cumm resulting in liberation of inteleukin-6 cytokines and tumor necrosis factor alpha. Cardiac enzymes are raised. Serum amylase mut be invetigated in all cases with upper abdominal pain with vomiting and tachycardia. Serum potassium is raised in early hypertensive phase. In a few cases there is reduction in serum calcium level. If facilities are available serum insulin detection is helpful for further research. Chet X-ray shows bilateral batwing, patchy or intertitial pulmonary edema. At times secondary respiratory infection in form of pneumonitis is often seen in a hospitalized patient recovering from pulmonary edema. On 4th day, mild cardiomegaly is seen in a case recovering from pulmonary edema.

Electrocardiography changes
Hyper-acute tented T waves, bradycardia, irt degree heart block, transient ventricular and atrial ectopics, runs of ventricular VPC's can be observed and detected if case is monitored by cardio scope. PQRST or T waves alternans, acute myocardial infarction like pattern, sinus tachycardia with ST segments depression, left anterior hemi-block, bundle branch block are also seen. Many times left bundle branch block with marked tachycardia is confused for ventricular tachycardia. In such a situation the bundle branch block is tachycardia dependent. Complete heart block, low voltage, widened QRS complexes with tachycardia carries poor prognosis. Prolonged QTc interval (0.50-0.60 seconds) with broad base and round top T waves with bradycardia with hypotension are seen 12-24 hours after ting in a hospitalized patient. Mot early changes in ECG such as left axis deviation, Tented T waves and ST segment elevation in lead I and AVL should be closely monitored for possibility of development of acute pulmonary edema.

Pathophysiology
The clinical manifetations, pathological lesions and electrocardiographic changes are due to sudden massive liberation of catecholamines in to circulation similar to pheochromocytoma. Cool extremities with severe vasocontriction (like Raynaud's phenomenon), dilated pupils, cold extremities, inhibition of insulin secretion, hyperglycemia, acute myocardial infarction like pattern (spasm of pericardial coronary vessels), pulmonary edema are due to alpha-1 receptors timulation due to catecholamine excess. Scorpion venom increases the membrane permeability to sodium by opening the voltage sensitive sodium channels, which is accompanied with calcium entry, and blockade of calcium activated potassium channels resulting in relative hyperkalemia. Hyperkalemia further induces the release of catecholamines. Stimulation of alpha-receptors causes potassium elux from the liver. Hyperkalemia, hypocalcaemia can explain the electrocardiographic changes-tall T waves, prolonged QTc and ST segment changes. Initial short lating hypotension is due to hypovolaemia, peripheral cholinergic and central vagus timulation. Delayed long lating hypotension occurs as result of vasodilatation and depleted catecholamines. Pulmonary edema in scorpion ting is of hemodynamic origin and is related to severe impairment of left ventricular sytolic function. Local pain is due to sodium channel activation and timulation of C ibers with local liberated protaglandins.

Management
No ting should be taken as benign unless observed for 24 hours irrespective of species of scorpion involved.

Local pain
Cut at the ting site and tourniquet is not advisable. Mild, tolerable pain can be abolished by application cold or ice packs over the site of ting. Severe excruciating pain is transiently relieved by local anethesia (xylocaine without adrenaline). Repeated injections of local anethesia are often required for pain relief. It is observed that the reappearance of pain after initial local xylocaine is much severe than what was before the irt injection. This is because of rapid tolerance to xylocaine and repeated injection timulates the inlammation and in an inlamed tissue the action of xylocaine is blunted. Hence simultaneously oral diazepam and NSAID with irt initial dose of xylocaine can give prolonged relief of pain. However

injection of emetine hydrochloride exactly at the site of ting gives prolonged relief from pain, but it is not available and moreover it is cardiotoxic and one need to be careful while injecting because ting is often over the thick skin of sole and many times while injecting it the drug may suddenly be lushed back due to dislodged needle and piton from syringe and enter in the eyes of a person injecting it. Being severe tissue irritant, it causes corneal edema and irritating injury to cornea (author himself sufered of

Correction of dehydration

Dehydration due to vomiting, excessive salivation, and profuse sweating should be corrected by continuous vigorous oral rehydration solution. This helps to correct initial hypotension and shock. Intravenous crytalloid solution or hydration by nasal tube may be necessary in a confused, agitated child. Fluid replacement mut be corrected since hypovolemia is one of the proposed mechanisms of shock syndrome in scorpion ting. Electrolytes imbalance should be corrected. Calcium can be replaced by encouraging excessive milk consumption. Grape juice may be avoided as it may enhance the prolonged the QTC interval

Scorpion antivenin

Scorpion antivenin is available in India. Though it is speciic antidote to venom action but in case of mesobuthus tamulus ting antivenin, it does not prevent or reverse the cardiovascular morbidity and mortality. Moreover it is not free from anaphylaxis. The half-life time of antivenin is longer than venom. In animals no beneicial efects of antivenin are observed if it is injected more than 15 minutes after injecting the venom. It does not counteract the venom induced autonomic torm. In our series, mesobuthus antivenin did alleviate the cardiovascular morbidity and mortality. 21 severe scorpion ting cases aging 3-56 (average22) years were reported within 30 minutes to 21 hours (average 3.5) after ting. All of them had clinical manifetations suggetive of autonomic torm. They received scorpion antivenin by intravenous route and were closely observed for clinical outcome. Out of these, 12 had persitent raised blood pressure, 8 developed pulmonary edema, of which 2 had massive life threatening pulmonary edema, 1 had hypotension with tachycardia and 2 died. Recently it has been reported that scorpion antivenin is no better than placebo. Venom is poor antigen hence it is diicult to prepared potent antivenin. Scorpion venom acts indirectly by releasing auto-pharmacological subtance in to circulation.

Prazosin

Alpha-receptors play vital role in the pathogenesis of cardiac failure and pulmonary edema due to scorpion ting. Prazosin is a selective alpha-1 adrenergic receptor blocker. It dilates veins and arterioles, there by reducing pre-load and left ventricular impedance without rise in heart rate and renin secretion. It also inhibits sympathetic outlow in central nervous sytem. It is phospo-dieterase inhibitor and as a result of this action it enhances cGMP accumulation, which is one of the mediator of nitric oxide synthesis. It enhances insulin secretion, which is inhibited by venom action. Thus its pharmacological properties can antagonize the haemodynamic, hormonal and metabolic efects of scorpion venom action. It can be adminitered by oral route. Prazosin is simple, scientiic pharmacological and physiological antidote to scorpion venom actions; moreover it is free from anaphylaxis. Prazosin should be given in a dose of 125-250 microgram in children and 500 microgram in adults and should be repeated three hourly until there are signs of clinical improvement in tissue perfusion such as warming of extremities, increase in urine output, appearance of severe local pain at the site of ting which was absent or tolerable on arrival, disappearance of parathesias, reduction or improvement in heart rate and pulmonary edema, reduction in hypertension or improvement in blood pressure in case of hypotension without hypovolemia, reduction or disappearance of murmur and earliet mot important subjective feeling of better. This is because the drug has 1000 times ainity towards the activated alpha-1 receptors. Then dose is to be repeated six hourly till extremities became dry and warm. If the initial dose has been vomited (one should see the vomit carefully), it should be repeated. In a confused, agitated, non-cooperative child, prazosin should be adminitered by nasal tube after giving intravenous diazepam **Prazosin is life saving drug hence attending doctor himself should adminiter the drug to the hospitalized patient and he should clinically conirm by noting the signs and symptoms that drug is absorbed in circulation and tarted acting.** Firt dose phenomenon is rare or avoided by adminitering minimum initial dose of prazosin. However due care should be taken to avoid potural fall in blood pressure. Children should not be lifted. Potural hypotension should be treated by giving head low position and intravenous luid.

Pulmonary edema Is a life threatening time limiting emergency often fatal and needs rapid intervention. Patient should be given propped up position, intravenous aminophylline 5mg/kg diluted in dextrose given as a slow bolus to counter the associated broncho-

9. *Bawaskar HS and Bawaskar PH. Clinical proile of severe scorpion envenomation in children at rural setting. Indian pediatrics 2003;40:1072-75.*

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