Scorpion Bite Atypically Presenting As Acute Transverse Myelitis And Sub Arachnoid Hemorrhage: A Case Report And Review

Arti Muley*, Dr. Harshalsinh S. Mahida, Kinjal Patel****

*Professor, Department Of Medicine, PIMSR, Parul University, Vadodara, Gujarat, India ,**Resident, Department Of Medicine, SBKS MIRC, Vadodara, Gujarat, India.

Abstract: Scorpion bite is an important health issue as it has been reported that about ten persons are killed by a venomous scorpion for each killed by a venomous snake. Scorpion venom may be cardiotoxic, hemotoxic, nephrotoxic or even neurotoxic. It acts on the autonomic nervous system producing parasympathetic and sympathetic manifestations. However, few have reported sub arachnoid haemorrhage and transverse myelitis occurring due to scorpion venom. Case Report: We are reporting a case of 50 year old male who presented three days after an episode of scorpion bite with paraplegia and inability to pass urine and stool due to transverse myelitis and subarachnoid hemorrhage. He was investigated and treated accordingly. Clinical improvement was seen within ten days after the initiation of therapy. Conclusion: **S**corpion sting, though rarely may present as SAH and transverse myelitis which are reversible and easily treatable. Clinical Significance: As scorpion bite is treatable, having high index of suspicion for scorpion sting in patients of SAH and acute transverse myelitis in whom the cause of their clinical features could not be recognised may help in improving the outcome considerably in these cases.

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Key Words: Scorpion Sting; Transverse Myelitis; Subarachnoid Haemorrhage

Author for correspondence: Arti Muley, Professor, Department Of Medicine, PIMSR, Parul University, Vadodara, Gujarat, India E-Mail: muleyarti40@gmail.com

Introduction: Scorpions are arthropods from arachnid family which have a tendency to bite at night. In warm climate, they remain hidden[1]. Worldwide incidence and mortality projected due to scorpion bite are about 1.2 million/year and 3250 deaths/year respectively. It has been estimated that about 10 persons are killed by a venomous scorpion for each killed by a venomous snake[2].

Scorpions mostly attack in defence, hence scorpion stings are usually accidental. The stings most commonly occur when a person unintentionally steps on a scorpion or reaches under wood or rocks[3]. The scorpion venom contains various toxins which may be cardiotoxic, hemotoxic, nephrotoxic or even neurotoxic and thus exhibits a wide range of clinical manifestations like excess motor activity leading to hyperthermia, metabolic acidosis, pulmonary fluid overload, respiratory failure, sterile rhabdomyolysis, coagulopathy, pancreatitis, multiple organ failure, cerebrospinal fluid pleocytosis and neurotoxicity[4].

The majority of scorpion bites with neuromuscular toxicity are minimally symptomatic with no to minimal inflammation locally and local pain sometimes. If present, symptoms may begin immediately and become most severe within 5 hours which can be life threatening in upto 30% of patients stung by Centruroides or Parabuthus species. Para-sympathomimetic effects due to neuromuscular involvement from scorpion bite may lead to cranial nerve dysfunctions presenting as dysphagia, increased oral secretions and drooling, tongue fasciculations, combination of bulbar neuromuscular dysfunction and slurred speech, abnormal eye movements and blurring of vision[5].

Scorpion bites may also cause somatic skeletal neuromuscular dysfunction presenting as restlessness, fasciculations, shaking and jerking of the extremities, alternating opisthotonos (arching of the back) and emprosthotonos (tetanic forward flexion of the body), salivation, vomiting, bronchoconstriction, diaphoresis, tachycardia and urinary retention[6].

However, there are very few reports of transverse myelitis and subarachnoid hemorrhage developing as manifestation of scorpion bite. We are presenting a rare case of scropion bite which induced unstable angina, sub arachnoid hemorrhage and transverse myelitis simultaneously.

Case Description: A 50 year old right handed male, watchman by occupation, chronic smoker and chronic alcoholic was brought to the emergency with chief complaints of weakness in both lower limbs and bowel bladder incontinence since three days. The patient was apparently alright before four days when in the evening he was bitten by a scorpion on the dorsum of left foot below great toe followed by a burning pain at the site of bite which lasted for some hours and then subsided. It was not associated with any swelling, itching or redness. After 90 minutes of bite he developed chest pain and throbbing frontal headache.

The chest pain was sudden in onset, retrosternal, constricting but non-radiating, for which he was taken to some hospital where he was admitted and routine investigations and ECG were done. ECG showed tall T waves in both limb leads and precordial leads, for which low molecular weight heparin and nicorandil were started.

Oral rosuvastatin 40 mg, aspirin 325mg and clopidogrel 300mg were given stat to be followed by aspirin 150mg, clopidogrel 75mg and spironolactone 25mg plus torsemide 10mg daily.

At that time he also had low backache which was severe in intensity, radiating to both lower limbs.

The pain was electric shock like and used to aggravate on change of posture. After a few hours he developed left lower limb weakness and was not able to pass stool and urine.

On the second day of admission he noticed that he was not able to move his right lower limb also and observed numbness and decrease in touch sensation in both lower limbs associated with feeling of belt like tightening over abdomen just above the umbilicus.

At the same time he also had difficulty to sit from supine position without support. In view of persistent symptoms he took discharge from the hospital and went to another hospital where at presentation, his blood pressure was 160/90 mmHg while rest of the vital parameters were within normal limits. Here again some blood tests and ECG were done.

ECG was same as the previous one. After that he underwent coronary angiography which was normal.

Hence a diagnosis of unstable angina was made and the dual anti platelets and anticoagulants were continued. Meanwhile, the lower limb weakness persisted although it was not progressive. For the same, the patient was brought to our hospital for further management.

He had no history of seizures, blurring or loss of vision, deviation of angle of mouth, slurring of speech, fever, trauma or any preceding history of diarrhoea, nausea or vomiting. There was no history of similar complaints, hypertension, diabetes, ischemic heart disease, stroke, tuberculosis or blood transfusion in the past.

Family history was not remarkable. He was vegetarian with normal appetite and adequate sleep but had bowel and bladder incontinence. He was chronic smoker and alcoholic (1 bundle of cigarette/day and 100 ml of country liquor/day since last 20 years).

On examination at presentation, he was afebrile, pulse rate was 86 beats/ min, regular, blood pressure was 118/70 mmHg and respiratory rate was 18/min abdominothoracic type with a normal BMI. There was no sign of pallor, icterus, cyanosis, clubbing, lymphadenopathy or edema.

No abnormality was detected in respiratory, cardiovascular and per abdominal examination. Higher mental functions, speech and cranial nerve examination were also normal.

Nutrition was normal. Tone was normal in both upper limbs while in both lower limbs it was decreased. Power was normal in both upper limbs in all joints for all movements while in both lower limbs it was 1/5. Sensations (both superficial and deep) in both upper limbs were normal but all the sensations were absent below umbilicus and in both lower limbs.

Deep tendon reflexes such as knee jerk and ankle jerk were absent in both lower limbs. Biceps triceps and supinator jerks were +2 in both upper limbs.

Lab investigations showed only mild leucocytosis and mildly elevated SGPT (Table 1). ECG was suggestive of cor pulmonale.

There was LVH on 2D ECHO. CT and MRI of brain showed subarachnoid haemorrhage while MRI of DL spine was suggestive of transverse myelitis.

Hence a diagnosis of subarachnoid haemorrhage with transverse myelitis with cor-p due to scorpion bite was made. Based on the diagnosis, injectable methylprednisolone 1 gm daily was given for five days along with antibiotics, anti hypertensives and other symptomatic and supportive treatment. With the treatment, patient slowly retained bowel, bladder sensation and voluntary movements at ankle joints.

He was discharged on request on oral steroids, antibiotics, antihypertensives and antiplatelets with other supportive medications with advice to follow up.

Table 1: Lab Investigation

	20/7/2019	**23/07/2019**
Haemoglobin(gm%)	13.3	12.1
TLC (cells/cu.mm)	13600	12000
DLC (cells/cu.mm) (N/L/M/E)	80, 11, 4, 5	72/20/4/4
Platelet (lacs/cu.mm)	1.82	2.07
RBC (mill/µl)	5.41	5.50
Prothrombin time (control -14)	Test -17.8- INR -1.27	
APTT (control -30)	Test -30	
B.urea (mg%)	60	36
S.creatinine (mg%)	1.3	0.9
Total Bilirubin (mg%)	0.9(Direct-0.4, Indirect 0.5)	
SGOT (IU/L)	98	
SGPT (IU/L)	100	
S.Na^+(mmol/lit)	140	133
S.K^+ (mmol/lit)	4.0	3.4
S.Cl^- (mmol/lit)	103	35
Urine routine	Albumin - +++ Sugar- absent Pus cell- 8-9 cell/ml Epithelial cell – 1-2cell/ml RBC- 2-3 cells/ml	
ECG	Changes of cor-p.	
HIV/HbSAG/HCV	Negative	
CSF study	pH-7.0 sugar – 25mg/dl protein- 88 mg/dl ADA- 09 LDH – 625 u/l	
CSF (culture)	No organism detected	
USG (abdomen/pelvis)	No abnormality detected	
Chest X ray	Within normal limits	
CT brain plain	Hypodense subarachnoid hemorrhage noted in involving convexity sulci of bilateral parietal lobe, mild sub arachnoid hemorrhage also noted involving sulci of bilateral parietal lobe sulci in parafalcine location.	
MRI brain and head and Neck angiography	Subarachnoid hemorrhage is seen in bilateral parieto-occipital and superior frontal sulci. Few tiny diffusion restriction foci are noted in left centrum semiovale - small acute ischemic foci.	
MRI DL spine with whole spine screening	There is intramedullary hyperintensity signal in spinal cord at D7 to D10 suggestive of transerverse myelitis Other findings are :- Arachnoditis, Lumbar spondylosis, Cervical spondylosis	
2D ECHO	**18/07/2019**	**27/07/2019**

	Concentric LVH No regional wall abnormality LVeF = 45% Grade I LV diastolic dysfunction Mild LV systolic dysfunction Trivial TR/ Trivial MR No PAH/MS/AR/AS	Concentric LVH No regional valve abnormality LVeF = 60% Grade I LV diastolic dysfunction Mild TR/ Mild PAH No MS/MR/AR/AS

Figure 1 (A): ECG On 2nd day Of Scorpion Bite Showing Sinus Rhythym, Normal Axis And Tall T Wave (17/07/2019)

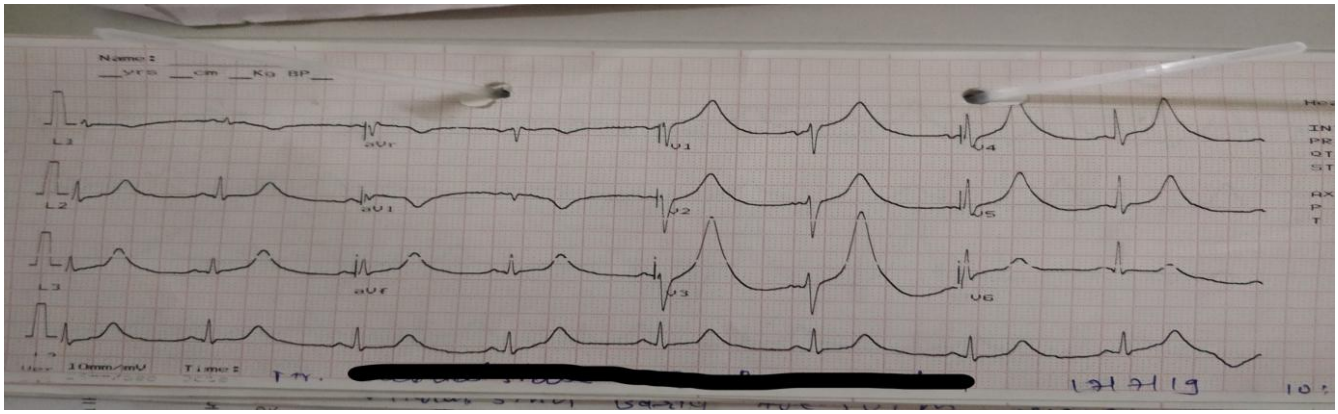


Figure 1 (B): ECG On 5thday Of Scorpion Bite Showing Sinus Tachycardia, Normal Axis, No ST-T Changes And "P" Pulmonale (20/07/2019)

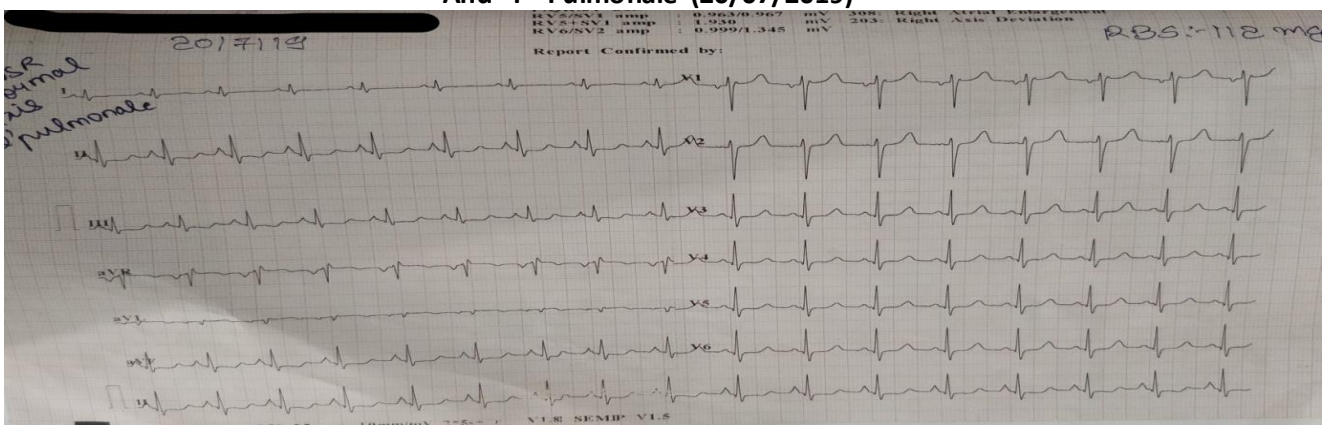


Figure 2(A): T2W Showing Transeverse Myelitis At D7-D10

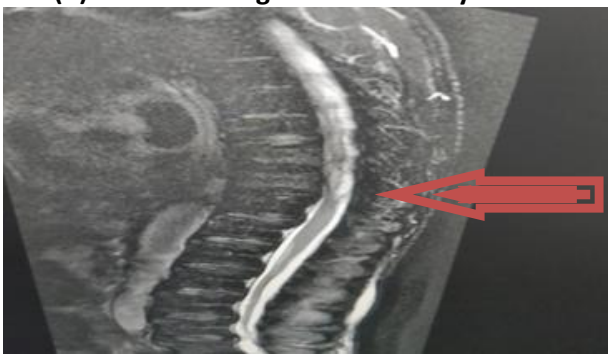
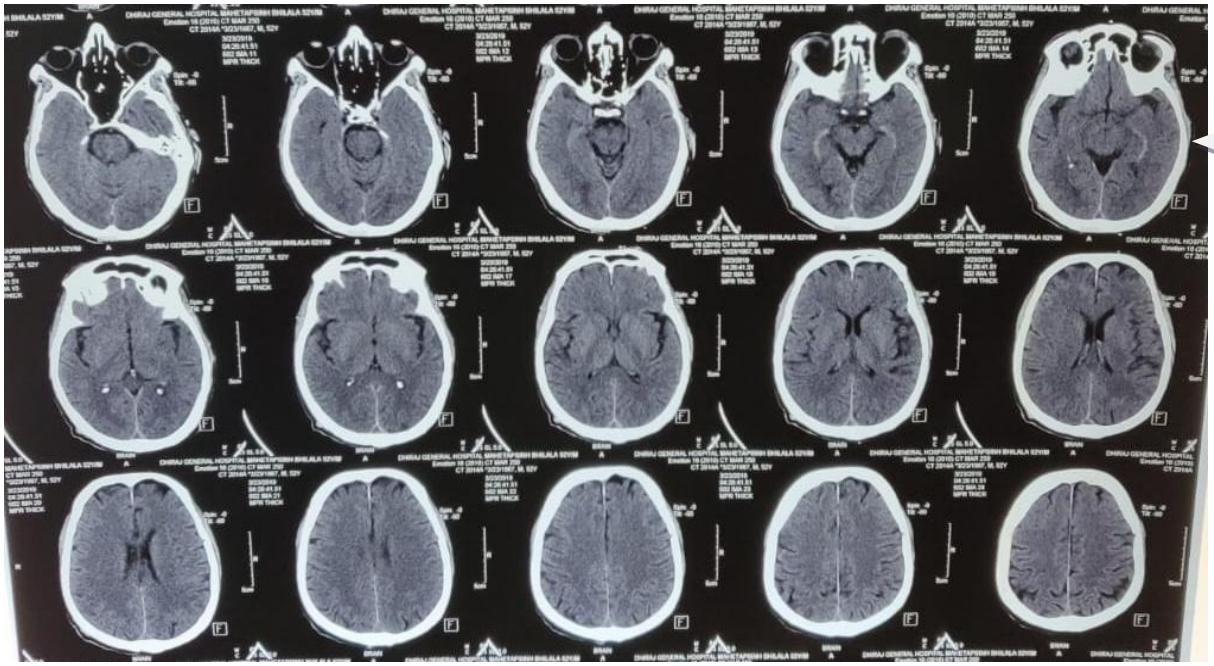


Figure 2 (B): CT Brain Plain Showing Subarachnoid Haemorrhage



Review: Scorpion stings are usually unintentional and most commonly occur on an extremity when the person unknowingly steps on the scorpion[3]. Actual envenomation occurs through stinging, not biting. The venomous scorpions have thin bodies and weak looking pincers. Their tail is thick and segmented with bulbous end (telson) that contains venom secreting vesicles with sharp semi curve stinger[7]. Maximum scorpion stings occur in rural areas in summer most commonly in the lower limb[8].

Scorpions inhabitat all continents except Antarctica. Around nine families with 1400 species of scorpions are known[9]. Out of these only the Buthidae family causes clinically significant neurotoxic envenomation[10]. At least 30 species can cause fatal stings[9-11].

Centruroides in the United States, Mexico, Central America and Parabuthus in South Africa are the most dangerous species[12-13]. The scorpion venom consists of several toxins like low molecular weight proteins, neurotoxins, aminoacids, oligopeptides, cardiotoxin, nephrotoxin, haemolytic toxin, phosphodiesterase, phospholipase A, hyaluroinidase, acetylcholine esterase, glycosaminoglycans, histamine, serotonin and proteins that inhibit protease[14]. Thus, it may cause widely variable clinical manifestations.

Main molecular target of scorpion neurotoxin are the voltage gated sodium channels and potassium channels including calcium channels.15 In human scorpion envenomations, alpha-toxins play an important part[16-18]. They prolong depolarization by inhibiting inactivation of sodium channels in neuronal membranes leading to membrane hyperexcitability, repetitive uncontrolled firing of axons[10] and release of neurotransmitters at synapses leading to excessive neuromuscular activity[19] and massive release of both sympathetic and parasympathetic neurotransmitters called as “Autonomic Storm”.

Scorpion toxin is cardiotoxic also and can have various effects on myocardium, which are usually transient. It can cause tachycardia, hypotension, hypertension, angina, myocardial infarction, arrhythmias and poor global myocardial contractility after 12-15 hours of sting, with low ejection fraction, decreased left ventricular performance, trivial mitral regurgitation, normal diastolic filling for five days to four weeks[20].

On ECG scorpion venom can cause low voltage, wide QRS complex, tachycardia, hemiblock and marked ST segment depression and T wave inversion which may persist for few weeks. Other changes which can be seen are peaked T wave known as ‘Ashoka T wave’ in V2-V6, ST segment elevation in lead I, aVL and increased QR duration and LVH. But all these changes are reversible[21-22].

SAH has rarely been reported in cases of scorpion sting. To the best of our knowledge, there is only one report of tranverse myelitis due to scorpion

sting till date. Rosenberg et al. in 1982 reported a case in which a 32 year old female developed sudden onset flaccid paralysis in both lower limbs 11 days after the scorpion sting, which got fully recovered with steroid therapy[23].

The clinical classification of the scorpion stings is adapted from an international consensus24 according to which the cases are classified into four grades.

Grade I: Only local pain and paresthesias at the sting site without local inflammation. While history of scorpion bite is typically present in adults, it may be absent in young children, hence the need for high index of suspicion in those presenting with unusual neurological symptoms.

Grade II: The distal symptoms radiate proximally in the extremity but may occur in contralateral limbs or may spread to cause generalized paresthesias. Children may present with unexplained agitation or inconsolable crying.

Grade III: May produce either cranial nerve or somatic skeletal neuromuscular dysfunction and autonomic dysfunction. The patient may have dysphagia, drooling of saliva, slurred speech, fasciculation of tongue, blurring of vision, restlessness, fasciculations, shaking and jerking of extremities, alternating opisthotonos and emprosthotonos and abnormal eye movements (involuntary, conjugate, slow and roving). Chaotic multidirectional conjugate saccades resembling opsoclonus and unsustained primary positional nystagmus may also be seen.5 The clinical features due to parasympathetic nervous system include bradycardia, increase in secretion like salivation, bronchorrhea, increase in gastric secretion, hypotension and priapism while activation of sympathetic nervous system causes tachycardia, hypertension, sweating, arrhythmias, myocardial infarction, pulmonary edema etc[25-27].

Grade IV: May manifest as hyperthermia, sterile cerebrospinal fluid pleocytosis, pulmonary edema, respiratory failure, metabolic acidosis, rhabdomyolysis, pancreatitis and multiple organ failure especially in children.4 Rarely pancreatitis is also seen but is typically transient. Scorpion venoms generally do not produce coagulopathy or other significant hematologic effects, although disseminated intravascular coagulation has been reported[28].

After the sting, symptoms may progress from minimal to maximal within 5 hours; while in infants they may progress within 15 to 30 minutes after being stung.29-30 Even without antivenom therapy, Grade III or IV envenomations improve symptomatically within 9 to 30 hours[10,31]. However, pain and paresthesias may persist for up to two weeks.

Scorpion stings must be differentiated from spider bite, tetanus, botulism, neuroblastoma, organophosphate poisoning, drug abuse, strychnine poisoning, meningitis and status asthmaticus.

Abnormal eye movements seen with scorpion stings are not seen in cases of spiderbite, tetanus, strychnine poisoning, meningitis and status asthmaticus[6,32]. In addition, black widow spider bites frequently produce a characteristic halo lesion at the site. Botulism does not cause hypersalivation, opsoclonus, fasciculation, or painful skeletal muscle contractions. Neuroblastoma manifests with – Opsoclonus-myoclonus-syndrome (rapid, dancing eye movements, rhythmic jerking and/or ataxia) and does not have hypersalivation, acute onset of cranial nerve deficits or skeletal muscle effects. Organophosphates frequently cause paralysis and true seizures with loss of consciousness, which is not seen with scorpion envenomation. Drug abuse usually presents with delirium also and does not cause cranial nerve dysfunction.

Paresthesias, skeletal muscle abnormalities and cranial nerve abnormalities are not seen in patients with foreign bodies and status asthmaticus[6].

Other than absence of abnormal eye movements, a history of an infected wound (tetanus), exposure to a precipitating agent (dystonic reaction), or strychnine ingestion can facilitate making these alternative diagnoses[6,33,34].

Management: Sting site should be cleansed and tetanus prophylaxis should be given to all patients irrespective of the severity. Other than this, mild to moderate local pain and/or paresthesias may require only NSAID or oral opioid medications depending upon the degree of pain. For severe local or remote pain and/or paresthesias, regional anesthesia and short-acting intravenous opioids may be used followed by four hours observation to ensure no further

progression of symptoms[33-34]. Patients can be discharged once they start taking orally, pain is controlled with oral medicines and have no progression of symptoms. Patients with Grade III or IV severity, should receive intravenous scorpion-specific antivenom as per the WHO recommendations for Centruroides and Parabuthusspecies. However, antivenom is not life-saving but only reduces the duration of suffering and hospitalization. Anaphylaxis and serum sickness may occur occasionally with the venom and should be duly treated[35].

Other supportive interventions which may be required include frequent suctioning of oral secretions, endotracheal intubation, close monitoring for and treatment of myocardial ischemia and/or acute decompensated heart failure in patients at risk. If antivenom is not used, intravenous benzodiazepines (BZD) may be used for sedation and to treat muscle spasticity3, however they should be used carefully or avoided if antivenom administration is planned as antivenom reverses the excitatory effects of the scorpion venom, hence previous administration of BZD may result in over sedation and requirement of ventilation[35].

Reducing small cracks and crevices in homes, checking clothing and shoes, for scorpions before wearing, removing unnecessary ground cover and debris to reduce potential nesting places and spraying insecticides around the home can be effective in reducing incidence of scorpion stings[36].

Conclusion: Scorpion sting resemble number of diseases. An apparently healthy patient developed unstable angina, headache and bilateral lower limb weakness with bowel and bladder involvement after the scorpion sting. With high degree of clinical suspicion of transverse myelitis neuro imaging study was done which backed our clinical suspicion thus patient was treated with intravenous corticosteroid and clinical improvement was seen shortly after the treatment. Thus it can be concluded that sub arachnoid haemorrhage and transverse myelitis though infrequent, can occur after a scorpion bite.

Clinical Significance: We are presenting this case as our patient had subarachnoid haemorrhage as well as transverse myelitis which are rare manifestations of a common problem that is scorpion sting. To the best of our knowledge, till date only one case of tranverse myelitis due to scorpion bite has been reported. This case highlights the fact that having high index of suspicion should be kept for scorpion sting in patients of SAH and transverse myelitis in whom the cause of their clinical features could not be recognised. As manifestations of scorpion bite are reversible and easily treatable, this may help in improving the outcome considerably in these cases.

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