Case Report

Uncommon presentation of scorpion sting at teaching hospital

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Abstract

Scorpion envenomation is a major public health problem in tropical and sub-tropical countries, especially in Africa, Middle East, Latin America, and India. Even though most of the scorpion envenomation are harmless, it is generally seen with a set of clinical features, such as pain, edema, numbness, and tenderness in the area of the sting but rarely have serious clinical sequelae with involvement of vital organ systems like cardiovascular system and respiratory system leading to fatal manifestations like acute pulmonary edema, acute heart failure, and acute respiratory distress syndrome (ARDS). Here we present a case of a 19-year-old village boy who developed myocarditis and cardiogenic shock following scorpion envenomation, which was successfully treated with vasopressors, non invasive ventilation, and other supportive care.

Keywords: Arachnid stings, Himalayan scorpion bite, myocarditis, venomous bites

Introduction

It has been estimated that around 2.3 million population are at a risk of scorpion stings out of which roughly 1.2 million people are exposed to scorpion sting annually worldwide.[1] Scorpion envenomation is a major public health problem in India. The morbidity and mortality due to scorpion envenomation remains significantly high especially in rural areas due to delay in hospitalization. In the Himalayan foothills, there is a common belief that scorpion sting is not lethal unlike snakebite and also there is a common belief that symptoms will subside after few hours by using some home remedies. This leads to delay in starting medical management for these cases.

Nearly 1,000 species of scorpion are known worldwide, which belong to six families. However, only the scorpion belonging to the family Buthidae, secretes neurotoxic venom that is toxic to human. Around 86 species of scorpion are found in India. Among them *Mesobuthus tamulus* (Indian red scorpion) and *Heterometrus swammerdami* (black scorpion) are of medical importance.[2]

The scorpion venom is a water-soluble antigenic complex, which is a composite mixture of neurotoxin, cardiotoxin, nephrotoxin, hamolysins, phosphodiesterases, phospholipases, hyaluronidases, histamine, and other chemicals. The primary target of scorpion venom is voltage-dependent ion channels. The cardiovascular toxic effect of the venom causing toxic myocarditis is by the reduction of Na, K-ATPase, and adrenergic myocarditis is by releasing adrenaline and nor adrenaline from neurons, ganglia, and adrenals, thereby increasing myocardial oxygen demand by direct inotropic and chronotropic effect on already compromised myocardial blood supply.[3]

The scorpion envenomation is common during monsoon and summer season. The clinical features at presentation are

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Figure 1: Indian red scorpion, picture captured by patient friend after killing the scorpion

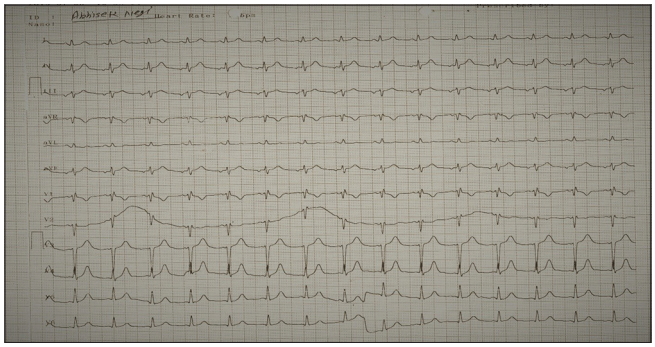

Figure 3: 12 Lead ECG (at the time of discharge) showing normal sinus rhythm with RBBB

pain, numbness, and tingling sensation at the sting site and the common complications associated are tachycardia, hypotension, myocarditis, and pulmonary edema. The cardiovascular effect is particularly prominent after stings by the Indian red scorpion (*M. tamulus*). The anticipation and close monitoring of complications and early prazosin therapy is required to reduce mortality and morbidity in scorpion envenomation.[4]

Here we present a case report of a 19-year-old male patient who developed myocarditis, pulmonary edema, and cardiogenic shock following scorpion envenomation. Who was successfully treated with inotropes, mechanical ventilation, and other supportive measures.

Case Report

A 19-yr-old male, resident of Kotdwar, Pauri Garhwal, Uttarakhand, was brought to emergency department with the history of scorpion sting (Figure 1 as clicked by attendants)over the right great toe six hours ago. Patient was out for a picnic with friends where he had scorpion sting. Initially patient had intense pain at the site of bite. He was taken to Kotdwar government hospital where he got first aid. This was followed by nausea, vomiting, profuse sweating, and abdominal pain. Subsequently

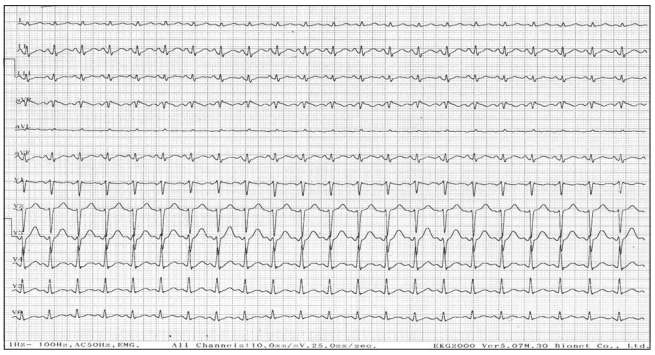

Figure 2: 12 Lead ECG (at admission) showing sinus tachycardia with RBBB

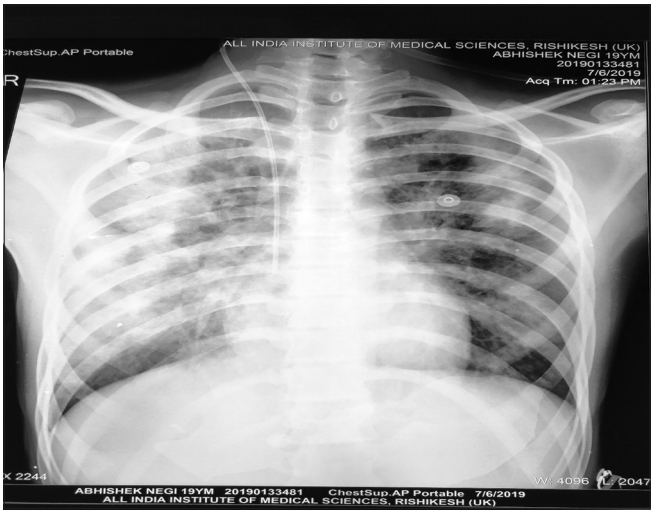

Figure 4: Chest Skiagram (AP view) on the day of admission: homogenous patchy infiltrates seen in bilateral lung fields

he developed shortness of breath and hypotension for which he was referred to higher centre (teaching hospital) for further management.

On admission, he was conscious and oriented. Patient's clinical examination revealed pulse rate of 116 beats/minute regular, respiratory rate 24 cycles/min and blood pressure 80 mmHg systolic, SPO2-70% on room air, extremities were cold, and he had cyanosis. On respiratory systemic examination bilateral fine crackles were heard. On cardiovascular examinations loud S3 gallop at apex andno obvious murmur was heard. His per abdomen and central nervous system examination were normal.

The electro cardiogram (ECG) of patient showed sinus tachycardia, his 2D Echo was suggestive of global left ventricular (LV) hypokinesia with moderate LV dysfunction (left ventricular ejection fraction (LVEF); 40%). Cardiac enzymes troponin I was positive. The patient was provisionally diagnosed with scorpion venom-induced myocarditis and cardiogenic shock. The patient was started on inotropic support in the view of cardiogenic shock. The patient also had the symptoms and signs suggestive of pulmonary edema which was managed with non invasive ventilation as the attendants of the patient refused

to give consent for mechanical ventilation. The patient was given other supportive measures in the form of inotropes, Table 1 shows the further hospital course and investigation, during the hospital stay. After 7 days of hospital stay, he was discharged in a hemodynamically stable condition. His 2 D Echo was repeated near discharge, which showed Normal LV function, LVEF; 55%, no pericardial effusion [Table 1].

Discussion

The case fatality due to acute pulmonary edema in scorpion envenomation during pre prazosin era (1961-1983) was found to be 25–30% in western India. Since the use of prazosin (1984), the incidence of mortality has reduced to 1% and case fatality in children has also reduced from 13% to 3% with the use of prazosin.[5]

Scorpion envenomation is usually a benign condition with a good prognosis. However, occasionally fatal complications like pulmonary edema, severe myocarditis, and cardiogenic shock can occur. The cardiac dysfunction in scorpion envenomation is possibly due to adrenergic myocarditis, toxic myocarditis, and

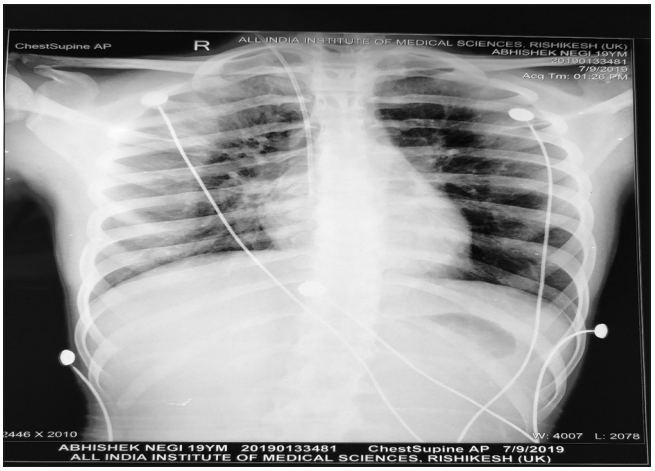

Figure 5: Chest Skiagram (AP view) on day 4 of in hospital care: homogenous patchy infiltrates seen in picture 5 are not seen on present picture

Table 1: sequential investigational report during hospital stay

DAY	Hemodynamic parameters & Investigation	Treatment Given
01	BP;70/50 mm of hg PR;126bpm, RR;28cpm Spo2;70% in room air Investigation: CBC: HB/TLC/DLC/Platelets; 15.2/18370/N86%,L9.7%, E3.6%/2.46lakh Urea/Creatinine;42/1.07 Tot. Bilirubin/Dir. Bilirubin; 0.7/ 0.26 SGOT/SGPT;74.8/45.8 CPK-MB/C-Trop-I; 137(0.25IU/l)/13.75(<1.5ng/ml) 2D-Echo: Global LV hyperkinesia, LVEF;45%, Minimal pericardial effusion seen. ECG: Figure 2 Chest X-Ray -Figure 4. ABG: Ph/Po2/Pco2/Hco3;7.41/47/26/17.1	Inj Noradrenaline (10mcg /min) InjDobutamine(1.5mcg/kg/min) NIV-BPAP(IPAP/EPAP:14/8) Inj pantoprazole 40mg iv od
02	BP;100/60 mm of hg PR;118bpm, RR;24cpm Spo2;94% on BPAP CPK-MB/C-Trop-I; 85(0.25IU/l)/23.24(<1.5ng/ml)	Inj Noradrenaline(10mcg /min) InjDobutamine(1mcg/kg/min) NIV-BPAP(IPAP/EPAP:14/8) Inj pantoprazole 40mg iv od Inj Furosemide 20mg iv BID
03	BP;100/60 mm of hg PR;110bpm, RR;20cpm Spo2;94% on BPAP CPK-MB/C-Trop-I; 46(0.25IU/l)/18.7(<1.5ng/ml)	Inj Noradrenaline (8mcg /min)-→slowly tapered off Inj Furosemide 40mg iv BID NIV-BPAP(IPAP/EPAP:12/6) Inj pantoprazole 40mg iv od
04	BP;100/60 mm of hg PR;110bpm, RR;20cpm Spo2;94% on BPAP Chest X-Ray -Figure 5	Inj Furosemide 40mg iv BID NIV-BPAP(IPAP/EPAP:12/6)--→intermittent Inj pantoprazole 40mg iv od
05	BP;100/60 mm of hg PR;110bpm, RR;20cpm Spo2;94% on oxygen CPK-MB/C-Trop-I; 30(0.25IU/l)/6.5(<1.5ng/ml)	Oxygen by nasal mask @ 2ltrs/min Supportive treatment
06	BP;100/60 mm of hg PR;110bpm, RR;20cpm Spo2;97% in room air CPK-MB/C-Trop-I; 20(0.25IU/l)/4.1(<1.5ng/ml) 2D-Echo: Normal LV function, LVEF;55%, no pericardial effusion ECG: Figure 3	Supportive treatment Discharged

myocardial ischemia. This was a probable cause of myocarditis in our case. Adrenergic and toxic myocarditis is due to the unopposed stimulation of alpha-adrenergic receptors by toxins, which in turn causes decreased insulin release, hyperglycemia, hyper kalemia, free fatty acid, and free radical accumulation, which are injurious to myocardium. Myocardial ischemia is due to the effect of excessive release of catecholamine's, cytokines, and neuro peptide Y on coronary vessels.[6]

The incidence of mortality was 35.71% in individual with scorpion envenomation with the evidence of the involvement of the myocardium.[7] Despite of refusal to mechanical ventilation by attendants of this patient, our patient was able to sale through the critical situation with inotropic support and non invasive ventilation. This gives a ray of hope in a way that young people may have a good organ resilience to tide over cardiovascular complications due to scorpion sting.

The management of scorpion envenomation depends upon the clinical condition. The mild symptoms like pain at the sting site should be managed with non steroidal anti-inflammatory drugs (NSAIDs) like aspirin. Individuals with mild systemic symptoms like tachycardia, hypertension, hyper secretory syndrome, and priapism should be treated with prazosin (30 mcg/kg po 6th hrly). Those with life threatening conditions like heart failure, cardiogenic shock, and pulmonary edema need ICU monitoring, vasopressor, and mechanical ventilator support.[8]

The scorpion envenomation is frequently seen in rural areas and first contact person in rural healthcare system will be rural general practitioners, rural physicians, and family physicians hence they should be aware of complications associated with scorpion envenomation and the early recognition of autonomic storm and to administer prazosin to prevent life threatening complications. (Discuss within the manuscript how this study is relevant to the practice of family physicians)

Take home message

1. Preventive measures like clearing the debris and trash from areas one inhabits, inspecting the boots and clothings before wearing, not to explore the places which one cannot see, using personal protective devices while working in farm should be followed.
2. The cardiac complications are common in Indian red scorpion envenomation. The underlying pathophysiology is due to excessive alpha receptors stimulation leading to myocardial dysfunction and acute pulmonary edema, hence Prazosin–an alpha adrenoreceptor antagonist can be used as antidote to venom action.
3. The clinical outcome of complication due to scorpion envenomation depends on time lapse between the sting and administration of prazosin for autonomic storm, hence early identification of autonomic storm and the administration of prazosin should be practised.
4. First contact health worker or physician must keep fatal complications in mind in patients with scorpion sting even if they are asymptomatic on initial presentation.
5. The area wise prevalence of types of scorpions and scorpion sting presentations must be documented for region specific management as per the type of venom in the particular area.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent forms. In the form the patients has given his consent for his images and other clinical information to be reported in the journal. The patient understands that his name and initials will not be published and due efforts will be made to conceal his identity, but anonymity cannot be guaranteed.

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Conflicts of interest

There are no conflicts of interest.

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