

Scorpion bite, a sting to the heart!

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Abstract

Scorpion bites are common in India. Usually, these bites are harmless but sometimes have serious clinical sequelae, including death. We report herein a case of scorpion bite with electrocardiographic abnormalities simulating early myocardial infarction. Pulmonary edema and congestive heart failure accompanied these electrocardiographic changes as well as serum cardiac markers. The etiology of cardiovascular manifestations in severe scorpion sting is related to venom effect on sympathetic nervous system and the adrenal secretion of catecholamines as well as to the toxic effect of the venom on the myocardium itself. It is a rare case of scorpion sting presented as myocardial infarction and heart failure, successfully treated with Intensive Care Unit care, noninvasive ventilation, vasopressors, and antiischemic treatment.

Keywords: Heart failure, myocardial infarction, scorpion bite

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Introduction

There are about 1500 species of scorpions worldwide, out of these 50 are dangerous to human. Among 86 species in India, *Mesobuthus tamulus* (Indian red scorpion) and *Heterometrus swammerdami* are of medical importance.^[1] Though local symptoms including severe pain and burning sensation at the site of sting are the most common manifestations, systemic complications can ensue.^[2] Cardiovascular manifestations are particularly prominent following stings by Indian red scorpion.^[3] Such bites infrequently have serious clinical sequelae, including myocardial infarction, acute pulmonary edema, cardiogenic shock, and even death. We present herein a case report with the clinical manifestations following scorpion bite mimicking acute myocardial infarction associated with acute pulmonary edema and congestive heart failure.

Case Report

A 14-year-old male resident of babaganj suffered a scorpion bite on right great toe. Within 3–4 h patient

starts having few episode of vomiting and loose stools, followed by chest pain associated with cold sweating. Blood pressure 70 mmHg systolic blood pressure was recorded by local medical practitioner and referred to K. G. M. U. Lucknow.

On admission, the patient complained of breathlessness, cough, and palpitation after 5–6 h of a scorpion bite. No significant past history and no other predisposed cardiac risk factor. Findings on clinical examination were as follows: Blood pressure 60 mmHg systolic blood pressure, pulse 140/min, regular and jugular venous pressure was raised; there was no cyanosis; the respiratory rate was 52/min. Examination of the respiratory system showed bilateral basal rales. Cardiovascular system examination revealed loud s3 gallop at apex, no murmur or pericardial rub, and no cardiomegaly. Central nervous examination patient is drowsy. Per abdominal examination was within normal limit except mild right hypochondrial tenderness.

Investigations

Laboratory investigations were as follows: Hemoglobin was 12.9 g/dL, white blood cell count was 30,470 cells/cmm, platelet count was 3.75 million/cmm, serum sodium was 149 mmol/L, serum potassium was 5.58 mmol/L; serum urea was 52.6 mg/dL, serum creatinine was 1.05 mg/dL, serum bilirubin was

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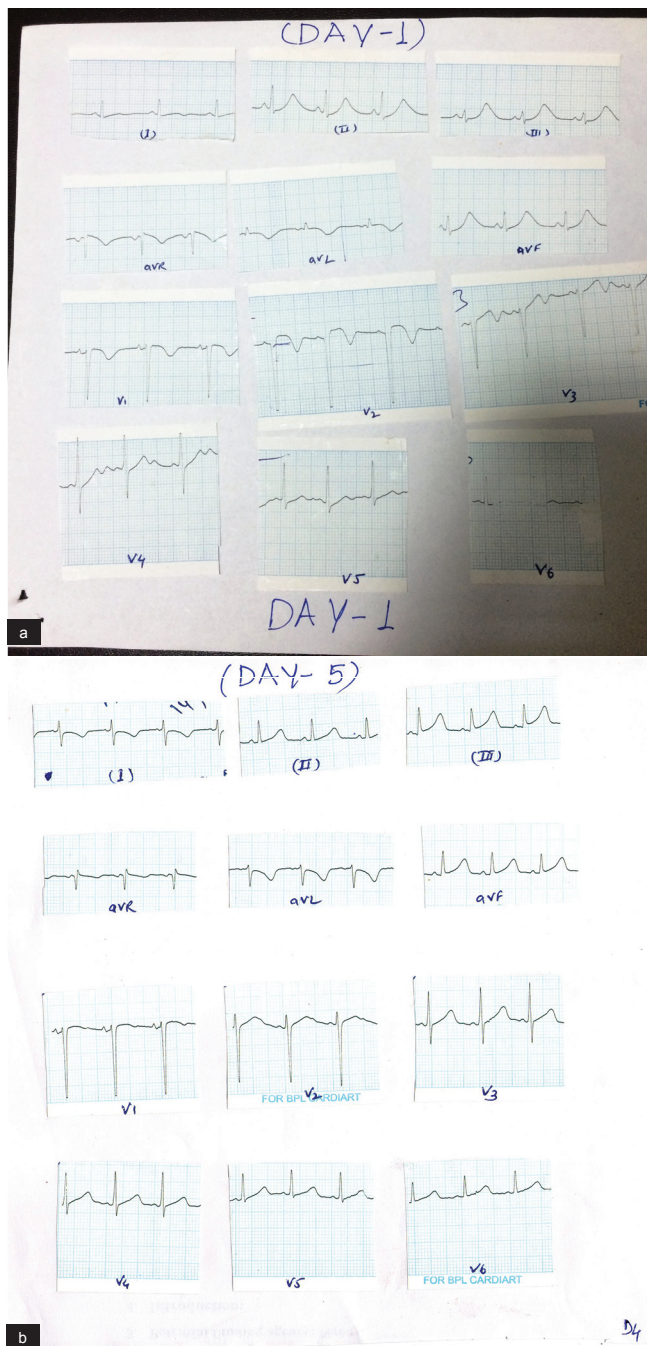


Figure 1: (a) Electrocardiogram (ECG) of the patient on the day of admission showing secondary ST- T changes and tachycardia, (B) ECG of the patient on day 5th showing T wave inversion in lead I and aVL

0.25 mg/dL, aspartate transaminase was 94.4 U/L, alanine transaminase was 27.1 U/L, serum alkaline phosphatase was 313 U/L, random blood sugar was 143.77 mg/dL.

Trop T was 0.382 ng/ml (0.000–0.014); ProBNP was 25845 pg/ml (0.000–125.0); CPKMB was 15.46 ng/ml (1.39–6.22).

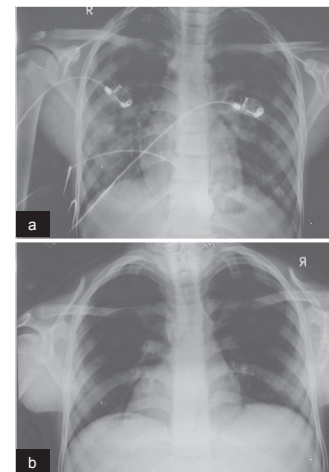


Figure 2: X-ray of the patient showing bilateral infiltrates suggested of pulmonary edema (a). X-ray of the patient on day 3 showing bilateral infiltrates suggested of pulmonary edema (b). X-ray of the patient on day 5th showing no infiltrates

Arterial blood gas analysis: pH 7.34, pCO₂ 34, pO₂ 50, sO₂ 82.4, serum lactate 2.9, serum bicarbonate 18.3, anion gap 27.

Electrocardiogram (ECG) taken on admission showed secondary ST-T changes and tachycardia [Figure 1a]. However, subsequent ECG on next day revealed normal sinus rhythm with ST segment elevation with concavity upward in lead II, III, aVF, V3, V4, V5, and V6, and T- wave inversion in lead I, aVL, V1, and V2; and Chest skiagram showed bilateral fluffy shadows suggestive of pulmonary edema [Figure 2a]. Echocardiogram demonstrates hypokinesia of the interventricular septum and inferior posterior wall with moderate mitral and tricuspid regurgitation and left ventricular ejection fraction 32%.

Differential Diagnosis

A differential diagnosis spectrum should be considered when chest X-ray showed bilateral fluffy shadow; congenital heart disease and rheumatic heart disease decompensated to congestive heart failure, atypical bilateral pneumonia.

Treatment

Patient was treated with intravenous dopamine, noradrenaline, dobutamine, frusemide and corticosteroids with oral aspirin, and nitrates, and noninvasive oxygen therapy in the form of continuous positive airway pressure with a nonrebreathing mask for type I respiratory failure.

Outcome and Follow-up

Patient was kept in Intensive Care Unit with oxygen supplementation and regular blood pressure, vital monitoring, and arterial blood gas monitoring. Patient's blood pressure increases gradually, repeat white blood cell count was 13910 cells/cmm, Trop T was 0.148 ng/ml (0.000–0.014); NT Pro BNP was 4571 pg/ml (0.000–125.0); CPKMB was 2.72 ng/ml (1.39–6.22); arterial blood gas analysis: pH 7.43, pCO₂ 36, pO₂ 83, sO₂ 98.0, serum lactate 0.9, serum bicarbonate 23.9, anion gap 15. Pulmonary edema subsided as Serial X-ray chest showed clearing up of the lung fields [Figure 2b], Serial ECG was taken over next 5 days, these showed persistence of q wave and T wave inversion in lead I and aVL [Figure 1b], and echocardiogram demonstrate mild mitral and tricuspid regurgitation and left ventricular ejection fraction 56%. Patient improved in 5–6 days. Patient was discharged, but unfortunately, the patient was lost in follow-up.

Discussion

The scorpion venom is a water soluble antigenic complex mixture of neurotoxin, cardiotoxin, nephrotoxin, hemolysins, phosphodiesterases, phospholipase, hyaluronidases, histamine, and other chemicals.^[2] The primary target of scorpion venom is voltage-dependent ion channels.

The venom produces both local as well as systemic reactions. Local reactions consist of itching, edema, and ecchymoses with burning pain.^[4] The cardiovascular manifestations comprise successively of giddiness, bradycardia, a fall of body temperature; restlessness and tachycardia; and finally pulmonary edema.^[5]

The venom can cause myocardial damage by several pathogenetic mechanisms:

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.^[6]

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 by direct inotropic and chronotropic effect on already

compromised myocardial blood supply.^[7]

Anaphylactic reaction

Release of allergenic proteins causes anaphylactic shock leading to hypotension with vasodilation and decreased of intravascular volume with reduced myocardial perfusion. Scorpion venom inhibits angiotensin converting enzyme, resulting in accumulation of bradykinin, which is implicated in the development of pulmonary edema.^[8]

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.^[9]

Valdivia *et al.* reported a series of 32 children with scorpion bite who developed cardiac complications. Among these 50% exhibited myocarditis, 12.5% had subclinical disease, 63% had observed ECG changes.^[10]

Levine *et al.* reported 19 fatalities due to wasps 1 due to bees, most of the deceased were older than 50 years and had underlying cardiovascular disease while previous reaction after insect stings were uncommon. Symptoms and death occurred within minutes after the sting. The autopsy finding was in most cases nonspecific.^[11]

In our patient finding mimicked acute myocardial infarction (including clinical symptoms, changes in ECG, elevated cardiac enzymes, regional wall motion abnormality in echocardiogram). Probably, coronary vasospasm associated with above-mentioned sequences (myocarditis, pulmonary edema) has precipitate acute myocardial infarction in our case. Acute myocardial infarction occurs very rarely after an arthropod envenomation. There are few cases of acute myocardial infarction due to bee or scorpion bite reported in the literature.^[11,12] Hence, it worth reporting this rare case.

Learning Points

1. Scorpion venom can have a potent cardiotoxic effect though rare but life-threatening, acute myocardial infarction and pulmonary edema can develop, after a scorpion bite
2. Any age group (as in this case a 14-year-old healthy male) can develop cardiac complication irrespective of previous health conditions
3. A routine serial ECG must be included in the

investigation to early detection of any cardiac manifestation and if required cardiac markers and echocardiogram helps in early diagnosis of acute cardiac complications like acute myocardial infarction.

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References

1. Erfati P. Epidemiology, symptomatology and treatment of buthinae stings. In: Bettini S, editor. *Arthropod Venoms: Hand Book of Experimental Pharmacology*. New York: Springer Verlag; 1978. p. 312-5.
2. Bawaskar HS, Bawaskar PH. Scorpion sting. *J Assoc Physicians India* 1998;46:388-92.
3. Bawaskar HS, Bawaskar PH. Indian red scorpion envenoming. *Indian J Pediatr* 1998;65:383-91.
4. Wallace JF. Disorders caused by venoms, bites and stings. In: Isselbacher KJ, Adams RD, Braunwald E, Petersdorf RG, Wilson JD, editors. *Harrison's Principles of Internal Medicine*. 9th ed. Johannesburg, London, Tokyo, etc.: McGraw-Hill International Book Co.; 1980. p. 924-5.
5. Mundle PM. Pulmonary edema following scorpion stings. *Br Med J* 1961;1:1042.
6. Yang HP, Chen FC, Chen CC, Shen TY, Wu SP, Tseng YZ. Manifestations mimicking acute myocardial infarction after honeybee sting. *Acta Cardiol Sin* 2009;25:31-5.
7. Rahav G, Weiss AT. Scorpion sting-induced pulmonary edema. Scintigraphic evidence of cardiac dysfunction. *Chest* 1990;97:1478-80.
8. Bawaskar HS, Bawaskar PH. Management of scorpion sting. *Heart* 1999;82:253-4.
9. Bahloul M, Kallel H, Rekik N, Ben Hamida C, Chelly H, Bouaziz M. Cardiovascular dysfunction following severe scorpion envenomation. Mechanisms and pathophysiology. *Presse Med* 2005;34:115-20.
10. Valdivia HH, Kirby MS, Lederer WJ, Coronado R. Scorpion toxins targeted against the sarcoplasmic reticulum Ca (2+) -release channel of skeletal and cardiac muscle. *Proc Natl Acad Sci U S A* 1992;89:12185-9.
11. Levine HD. Acute myocardial infarction following wasp sting. Report of two cases and critical survey of the literature. *Am Heart J* 1976;91:365-74.
12. Wagdi P, Mehan VK, Bürgi H, Salzmann C. Acute myocardial infarction after wasp stings in a patient with normal coronary arteries. *Am Heart J* 1994;128:820-3.

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