Int Cardiovasc Res J.2015;**9**(1):55-59.

International Cardiovascular Research Journal
www.ircrj.com

Acute Toxic Myocarditis and Pulmonary Oedema Developing from Scorpion Sting

Cem Sahin [1], Ethem Acar [2], Halil Beydilli [2], Kadir Ugur Mert [3], Fatih Akin [3], Ibrahim Altun [3,*]

[1] Department of Internal Medicine, Faculty of Medicine, Mugla Sitki Kocman University, Mugla, Turkey
[2] Department of Emergency, Faculty of Medicine, Mugla Sitki Kocman University, Mugla, Turkey
[3] Department of Cardiology, Faculty of Medicine, Mugla Sitki Kocman University, Mugla, Turkey

ARTICLE INFO

Article Type :
Case Report

Article History:
Received: 24 Jul 2014
Accepted: 11 Nov 2014

Keywords:
Diabetes
Renal Hypertension
Oxidative Stress
Rats

ABSTRACT

The majority of scorpion stings are generally seen with a set of simple clinical findings, such as pain, oedema, numbness, and tenderness in the area of the sting. However, occasionally events, such as toxic myocarditis, acute heart failure, acute pulmonary oedema, and Acute Respiratory Distress Syndrome (ARDS), which occur in scorpion sting cases are a significant problem which determine mortality and morbidity. The case presented here was a 38-year-old man who developed acute toxic myocarditis, acute heart failure, and acute pulmonary oedema following a scorpion sting on the 3rd finger of his right hand.

►*Implication for health policy/practice/research/medical education:*
This case report reminds us the very rare cause of dilate cardiomyopathy. The majority of scorpion stings are generally seen with a set of simple clinical findings, such as pain, oedema, numbness, and tenderness in the area of the sting. However, occasionally events, such as toxic myocarditis, acute heart failure, acute pulmonary oedema, and ARDS, which occur in scorpion sting cases are a significant problem which determine mortality and morbidity.

1. Introduction

Scorpion stings are widespread in tropical and subtropical regions. Scorpion venom is a poison with selective activity in mammals and vertebrates. Depending on the type of scorpion, its venom leads to different complications. As the toxin has a complex structure consisting of neurotoxic proteins, salts, acidic proteins, and organic components, it may cause hematological, neurological, and cardiovascular symptoms.

Cardiovascular toxic effects and acute pulmonary oedema are the most important life- threatening complications of scorpion stings (1). Cardiogenic shock and pulmonary oedema are responsible for a vast majority of deaths. Cardiac involvement generally occurs as impaired left ventricle systolic function. This impairment contributes to development of pulmonary oedema. The rapid increase of cardiac muscle enzymes and sudden deterioration of cardiac functions after a sting are seen in the acute damage which develops in myocarditis. This acute damage basically triggered by the venom results in adrenergic expression or the direct effect of the toxin on myocardial fibrils. Immediately following a scorpion sting, an autonomic storm is responsible for hypertension, tachycardia, pulmonary oedema, and shock (2).

The clinical findings of poisoning which develop as a result of a sting vary depending on factors, such as weight, age, and general health status of the stung person and the location of the sting. Young age, stings in the head and neck areas, stings in more than one area, and neurological complications, such as convulsions, have been mentioned as poor prognostic factors in cases of scorpion sting.

In the present study, the case was a 38-year-old male patient who presented with complaint of a scorpion sting and was determined to have acute myocardial damage, acute heart failure, and acute pulmonary oedema.

*Corresponding author: Ibrahim Altun, Mugla Sitki Kocman University, Faculty of Medicine, Department of Cardiology, Turkey, Tel: + 0252-2115174, *E-mail:* ibrahim_altun@yahoo.com

2. Case Presentation

Two hours after a yellow scorpion sting on the 3rd finger of the right hand, a 38-year-old male patient was evaluated in the emergency polyclinic for complaints of swelling in the finger, pain spreading under the armpit, facial spasms, discomfort, cold sweats, nausea, vomiting, and development of respiratory problems. In the history, it was learned that the patient had presented at another hospital when the symptoms started and as he had evident shortness of breath, oedema of the larynx was considered and treatment of subcutaneous adrenalin, intravenous (IV) antihistamine, IV steroid, and bronchodilator were applied. Following this treatment, as agitation, respiratory problems, and tachycardia had not improved, the patient had referred to our center.

The patient had no known underlying diseases, including cardiac diseases. In the examination, his general status was poor with a tendency for sleepiness and difficulty in cooperation. The patient could follow simple instructions and the Glasgow Coma Scale (GCS) was calculated as 12. The extremities felt cold, blood pressure was 90/60 mmHg, pulse was 120/min (radial), respiratory count was 42/min, body temperature was 37 °C, and oxygen saturation was 79% while receiving oxygen at 5 L/min via a nasal cannula. On the distal phalanx area of the 3rd finger of his right hand, there was redness in the area of the scorpion sting. On auscultation, widespread inspiratory fine rales were heard in the lower zones of both lungs. In the cardiovascular system examination, there was tachycardia and S3.

Blood count values were determined as follows: WBC: 29600/mm3, Hgb: 15.49 g/dL, Hct: 48.7%, Plt: 354000/mL, and Neu: 26400/mm3. Besides, glucose, creatinine, amylase, C- reactive protein, troponin-T, pro-BNP, D-dimer, CK-mb, myoglobin, SGOT, and SGPT values were observed to be high. The laboratory findings of the patient before and after treatment have been presented in Table 1.

In the Emergency Department, while the patient was receiving oxygen at 5 L/min via a nasal cannula, arterial blood gas values were determined as pH: 7.28, PCO2: 41 mmHg, PO2: 50 mmHg, HCO3: 19 mmol, and O2 saturation: 79%. On direct radiographs, widespread consolidated areas were seen in both lungs particularly in the lower zones (Figure1, Panel A). On electrocardiography (ECG) of the patient who had sinus tachycardia, no significant ST and T changes were observed.

In echocardiographic evaluation, global hypokinesis, EF 33% (Figure 2), and mild mitral regurgitation were observed. As the case had high cardiac enzymes and deteriorated left ventricle function, the diagnosis was made of acute toxic myocarditis which developed secondary to pulmonary oedema. Thus, the patient was admitted to the internal medicine intensive care unit.

2.1. Progress

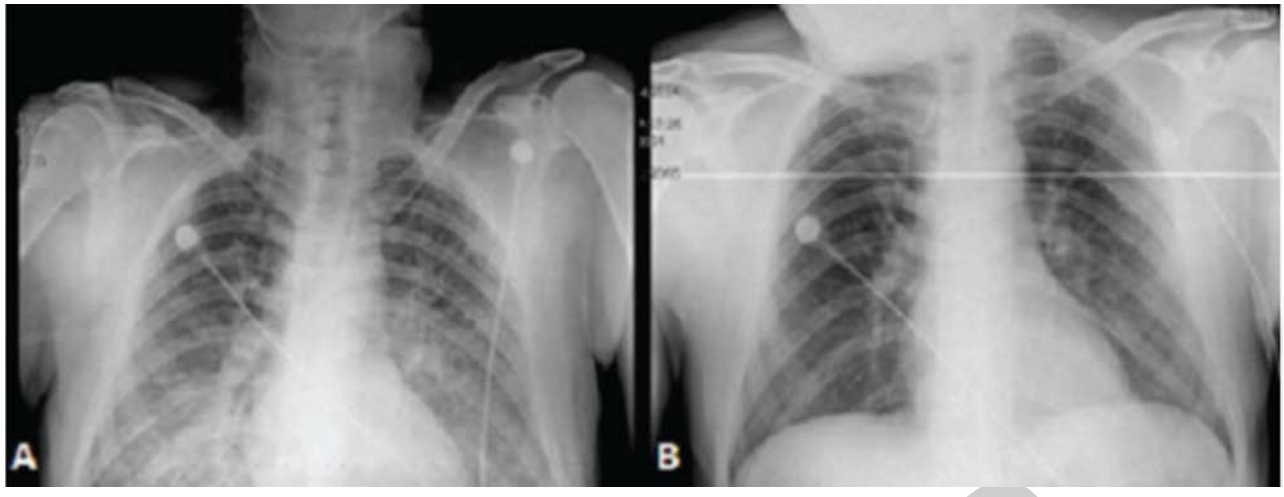
As the patient had deep hypoxia and pulmonary oedema, treatment of Non-Invasive Mechanical Ventilation (NIMV) was desired, but because the patient was greatly agitated, he could not tolerate NIMV. Hypoxia was continuing in the blood gases; therefore, the patient was intubated and attached to a mechanical ventilator with initial settings of volume control in Synchronized Intermittent Mandatory Ventilation (SIMV) mode, respiratory count of 12/min, Positive End Expiratory Pressure (PEEP) of 5cm H2O, tidal volume of 6ml/kg, and 70% oxygen. A midazolam infusion was also started for the agitation.

Treatment was applied by IV fluid electrolytes, empiric antibiotics, tetanus toxoid, antihistaminic, and 1 mg/kg steroids. Taking the necessary precautions against allergic reactions, serum anti-scorpion anti-venom was applied at intervals for 4 times. In the acute phase, prazosin was administered to prevent cardiac damage due to catecholamine discharge. IV nitroglycerine at a dose of 5 mcg/min was started for the acute cardiac failure and pulmonary oedema. As the arterial blood pressure was 90/60 mmHg, dobutamin (5 mcg/kg/min, iv infusion) was added to the treatment as positive inotropic support. The nitroglycerin and dobutamin infusion was continued for 48 hours. Spironolactone 50 mg and IV furosemide were

Table 1. Laboratory Results of the Case

	Pre-treatment	Post-treatment
Glucose (mg/dL)	222	98
Urea (mg/dL)	35	118
Creatinine (mg/dL)	1.3	0.7
Calcium (mg/dL)	8.5	8.7
Sodium (mmol/L)	145	140
Potassium (mmol/L)	3.9	4.3
LDH (IU/L)	382	43.6
AST (IU/L)	104	23
ALT (IU/L)	55	47
GGT (mg/dL)	62	81
ALP (IU/L)	146	122
Amilaz (IU/L)	301	79
CK-MB (g/dL)	10.97	1
Myoglobin (g/dL)	849	41
Troponin-T pg/mL	912	7
Pro-BNP pg/mL	2120	110
D-Dimer ng/mL	845	123
CRP (mg/L)	20.36	2.4

Figure 1. PA-x-ray Images of the Case Pre- and Post-Treatment



A, pre-treatment; B, post-treatment

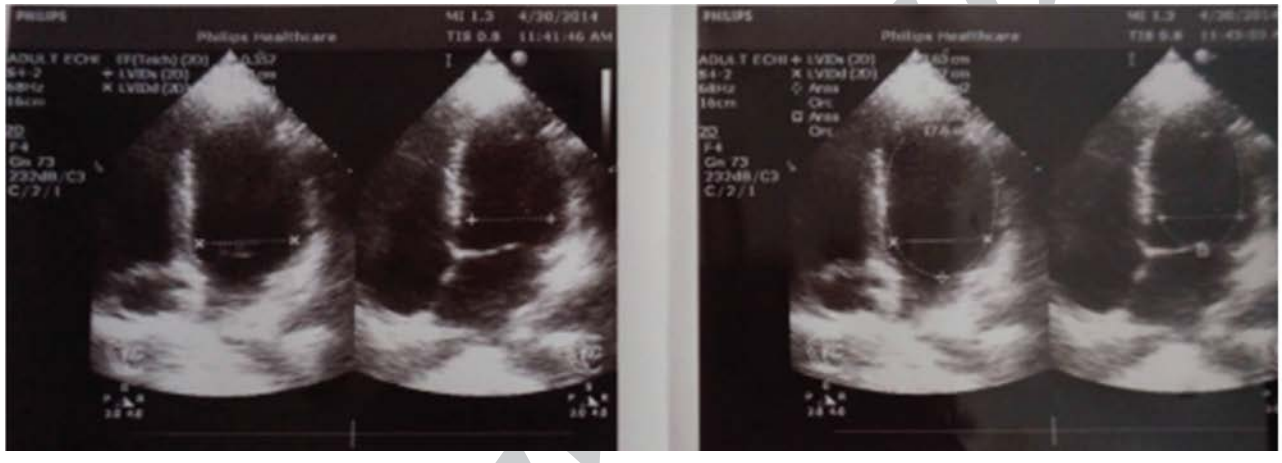


Figure 2. Echocardiogram of the Case Pre-Treatment

also administered daily to the patient. Daily monitoring was made of echocardiograph, ECG, cardiac enzymes, and biochemical parameters. After 24 hours, sinus tachycardia improved and following supportive treatment, the kidney and liver function tests were also observed to have recovered.

While monitoring the patient's blood gases, when oxygen saturation was over 90%, the Mechanical Ventilator (MV) oxygen support was gradually reduced. At the 48th hour, when the vital signs established, blood gases improved, MV support was minimized, and the patient was extubated. No reproduction was determined from the blood, urine, and tracheal aspirate cultures taken from the patient while he was in the intensive care unit. On the 4th day when the vital signs were normal, the patient was transferred from the intensive care unit to the clinic for continued observation.

On the echocardiograph applied 96 hours after admittance, the wall movements were seen to have returned to normal (EF 60%, Figure 3). The patient was discharged with low–dose oral beta-blocker and 50 mg spironolactone. On the 15-day follow-up examination, there were normal Echocardiography (ECHO) findings and physiological mitral failure on the echocardiograph. On myocardial perfusion scintigraphy, no findings related to any ischemic heart disease were determined.

3. Discussion

Viral infection lies in the etiology of a vast majority of cases of inflammation of the muscular wall of the heart, known as myocarditis. Scorpion sting is a rare cause of myocarditis. Clinically, a scorpion sting is generally seen with a set of simple findings, such as pain, oedema, numbness, and tenderness in the area of the sting. However, acute pulmonary oedema, ARDS, and acute heart failure have been shown to lead to serious respiratory and cardiovascular disorders, such as myocarditis, myocardial hypoperfusion, and arrhythmia. Cardiovascular toxic effects and acute pulmonary oedema are the most important life-threatening complications of scorpion stings (1).

Toxins found in scorpions, such as neurotoxin, haemolozine, aglutinine, leukocytoziline, coagulin, ferment, lecithin, and filoesterin, are responsible for the clinical condition which develops as a result of a scorpion sting (2). These toxins cause a set of local and systemic responses. Local responses include oedema, redness, and sensitivity, while systemic responses occur by scorpion serum opening Na channels from presynaptic nerve endings with the inhibition of Ca- associated potassium channels (2).

Although the pathophysiology of cardiac dysfunction

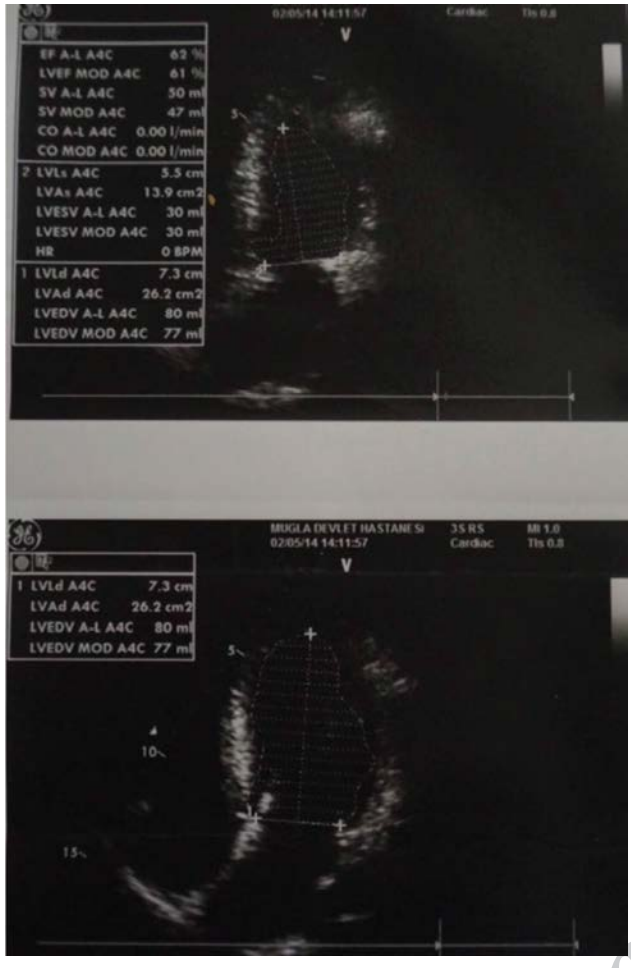


Figure 3. Echocardiogram of the Case Post-Treatment

developing secondary to a scorpion sting is still debatable, basically the direct toxic effect on cardiac fibrils of the toxin and the adrenergic expression triggered by the venom are held responsible. At the same time, it has been hypothesized that a contribution is made by some cytokines, such as neuropeptide Y and TNF-alpha, which are observed at increased serum levels in scorpion poisoning cases. All these mechanisms, separately or simultaneously, are thought to contribute to cardiac function (3).

Catecholamine mediated cardiac dysfunction is multifactorial. Catecholamine discharge results in increased heart rate, coronary spasm, and vasoconstriction occurring in the microcirculation, thus causing myocardial hypoperfusion and hypoxemia (3). At the same time, an increase in the intracellular concentrations contributes to direct toxic effects on myocardial cells and cardiac dysfunction.

Stimulation of alpha receptors leads to development of tachycardia, myocardial function disorder, pulmonary oedema, and circulatory disorders. If catecholamines are depleted in the late stage, hypotension, systolic function disorders, and pulmonary disorders will be observed (3).

In experimental animals, pulmonary oedema has been seen to develop following an IV injection of scorpion venom (4). The mechanism by which scorpion venom causes pulmonary oedema has not been fully clarified. However, it has been suggested that in the pathogenesis, the massive expression of catecholamine with a direct toxic effect of the venom on the myocardium and the venom-associated expressed cytokines may cause lung damage (3).

In the literature, it has been reported that the majority of the cases presenting with cardiovascular system symptoms have temporarily impaired left ventricle functions and cardiac functions have returned to normal after medical treatment (5). In these patients, it has been reported that temporary cardiac ischemia develops secondary to microvascular spasm and myocardial perfusion which develops as a result of the adrenergic discharge of acute cardiac dysfunction (3). Most cases recover clinically in a short time after a scorpion sting. However, in a study by Sundararaman et al., it was revealed that a scorpion sting was a risk factor in the long-term for development of idiopathic dilated cardiomyopathy (6).

Agitation is an important clinical parameter used in determination of the severity of a scorpion sting. In a study which evaluated the patients who developed pulmonary oedema following a scorpion sting, a significant relationship was found between agitation and development of pulmonary oedema (7).

Among the laboratory indicators, CK, CK-MB, LDH, and troponin values are used as indicators of cardiac effects. In cases of scorpion sting presenting with clinical cardiac symptoms, troponin-I may initially be determined at normal levels. In observation of these patients, troponin-I reaches its maximum level at 24 - 36 hours. In a study by Sagarad et al., high cardiac troponin levels in scorpion sting cases were shown to be a useful indicator in prediction of myocarditis as well as in planning for early treatment (8). In most cases, the clinical cardiac symptoms of increased cardiac enzymes and pathological ECG and ECHO findings have been reported to rapidly recover within 5 days. In the case reported here, CK, CK - MB, LDH, and troponin values which were high returned to the normal levels within 1 week after supportive treatment.

Although the ECG findings related to a scorpion sting are sometimes thought to be myocardial infarct, they are generally non-specific. In a previous study where cases of scorpion stings were followed up, ECG changes were determined in 71% of the cases and the most common rhythm impairment was atrium tachycardia. The ECG and ECHO findings of these cases were generally reversible (9). In the current case, the ECG change was tachycardia. Yet, improvement was observed in the ECG and ECHO tests repeated on the 5th day.

In addition to the cardiac effects of scorpion toxin, it can also cause systemic complications, such as impairment of renal functions, increased liver enzymes, hyperglycemia, impaired haemostasis tests, and thrombocytopenia. Scorpion venom may also cause pancreatitis due to excessive stimulation of the pancreas through cholinergic pathways. In the current case, during clinical follow-up, hyperglycemia, raised liver enzymes, impaired kidney function tests, and raised levels of amylase were observed. Following medical treatment, however, all these values returned to normal.

3.1. Treatment

For cases presenting with a scorpion sting, firstly the airway, respiratory, and circulation sufficiency must be

evaluated and the wound area must be cleaned by application of a tourniquet. In case there are systemic findings, repeated doses of scorpion anti-serum must be administered taking into consideration the precautions related to allergy. As supportive therapy for scorpion stings, fluid electrolyte support, tetanus toxoid, appropriate analgesia, and sedatives for agitated cases are recommended, as well. Besides, vitamin K and TDP can be used in the cases with hematological findings. Non-opiate analgesia has also been recommended to be administered for pain relief. For treatment of respiratory failure and pulmonary oedema, narcotic group analgesics, such as morphine, are not recommended as their synergic interaction with scorpion venom increases arrhythmia (10).

In cases of myocarditis which have developed associated with a scorpion sting, oxygen dopamine and dobutamin, sublingual nifedipin, digoxin and diuretic therapy are recommended for treatment of severe pulmonary oedema and heart failure. Adreneregic blockers, such as prazosin, have been shown to prevent the cardiac damage created by increased catecholamine in the acute period. However, angiotensin converting enzyme inhibitors have been reported to be of no benefit in the acute period (10).

3.2. Conclusion

Scorpion stings are frequently seen in our country and the stings of scorpions living in some regions may be fatal.

Although cases of scorpion stings are generally seen with simple local findings, it must be remembered that serious cardiovascular impairments, such as acute myocarditis, acute heart failure, and acute pulmonary oedema, may occur.

Particularly in cases with high levels of cardiac enzymes, it must be kept in mind that myocarditis can develop and the patients should be evaluated in this regard.

As scorpion sting is a risk factor in the long run for development of idiopathic dilated cardiomyopathy, cases should be included in a follow-up program.

Acknowledgements

There is no acknowledgement.

Authors' Contribution

All the authors contributed to all stages of the case report.

Financial disclosure

There is no financial disclosure.

Funding/Support

There is no funding/support.

References

1. Bahloul M, Ben Hamida C, Chtourou K, Ksibi H, Dammak H, Kallel H, et al. Evidence of myocardial ischaemia in severe scorpion envenomation. Myocardial perfusion scintigraphy study. Intensive Care Med. 2004;30(3):461–7.
2. Bhadani UK, Tripathi M, Sharma S, Pandey R. Scorpion sting envenomation presenting with pulmonary edema in adults: a report of seven cases from Nepal. Indian J Med Sci. 2006;60(1):19–23.
3. Cupo P, Figueiredo AB, Filho AP, Pintya AO, Tavares Junior GA, Caligaris F, et al. Acute left ventricular dysfunction of severe scorpion envenomation is related to myocardial perfusion disturbance. Int J Cardiol. 2007;116(1):98–106.
4. Freire-Maia L, Almeida HO, Cunha-Melo JR, Azevedo AD, Barroso J. Mechanism of the pulmonary edema induced by intravenous injection of scorpion toxin in the rat. Agents Actions. 1978;8(1-2):113–8.
5. Sezen Y, Guntekin U, Iscan A, Kapakli H, Buyukhatipoglu H, Kucukdurmaz Z. Rapidly improving, severe, acute myocarditis after a scorpion bite: an extremely rare complication and successful management. Am J Emerg Med. 2010;28(7):844 e3–5.
6. Sundararaman T, Olithselvan M, Sethuraman KR, Narayan KA. Scorpion envenomation as a risk factor for development of dilated cardiomyopathy. J Assoc Physicians India. 1999;47(11):1047–50.
7. Bouaziz M, Bahloul M, Hergafi L, Kallel H, Chaari L, Hamida CB, et al. Factors associated with pulmonary edema in severe scorpion sting patients--a multivariate analysis of 428 cases. Clin Toxicol (Phila). 2006;44(3):293–300.
8. Sagarad SV, Thakur BS, Reddy SS, Balasubramanya K, Joshi RM, Kerure SB. Elevated Cardiac Troponin (cTnI) Levels Correlate with the Clinical and Echocardiographic Evidences of Severe Myocarditis in Scorpion Sting Envenomation. J Clin Diagn Res. 2012;6(8):1369–71.
9. Diaz P, Chowell G, Ceja G, D'Auria TC, Lloyd RC, Castillo-Chavez C. Pediatric electrocardiograph abnormalities following Centruroides limpidus tecomanus scorpion envenomation. Toxicon. 2005;45(1):27–31.
10. Bayar N, Kucukseymen S, Yuksel IO, Arslan S. [Rapidly improving acute myocarditis after a scorpion sting]. Turk Kardiyol Dern Ars. 2013;41(7):629–32.