Case Report

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Asymmetrical and Late Onset of Pulmonary Edema Post Scorpion Sting: Case Report of Rare Manifestation

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Abstract

Scorpion bites are common in India and an important public health hazard in tropical and subtropical regions of India. Though generally bites are harmless, sometimes they can lead to serious sequelae including death. Herein we present a rare case of scorpion sting presenting as myocardial infarction manifesting in the form of asymmetric pulmonary edema after 24 hours of sting along with congestive cardiac failure, successfully treated with non invasive ventilation and inotropes. The etiology of the cardiovascular manifestations in scorpion sting is related to the venom effects on the sympathetic nervous system and the adrenal secretion of the catecholamines as well as to the toxic effects of the venom on the myocardium.

Keywords: Scorpion Sting; Pulmonary Edema; Congestive Cardiac Failure.

Introduction

Out of the 1000 scorpion species known worldwide only few are toxic to humans. Among the 86 species of scorpion present in India, Mesobuthus tumulus (Indian red scorpion) and Palmaneus gravimanus (black scorpion) 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 oedema and even death. We present here in a case report with the clinical manifestations following scorpion bite mimicking acute myocardial infarction.

Case Presentation

A 40 year old lady presented to the Emergency room with complaints of shortness of breath associated with profuse sweating since 1 hour prior to arrival. Her attendants gave alleged history of her being bitten by a scorpion in her right leg 2 days ago following which she had pain and swelling around the site of sting. She was taken to a local hospital for treatment where she was given intravenous fluids, hydrocortisone, and tablet prazosin but after two days she developed breathing difficulty, head reeling and sweating for which she was referred to this hospital for further management.

Her past history was not significant and she had no predisposing cardiac risk factors. Her initial blood pressure was 70/40 mm hg, heart rate 117 bpm, regular, oxygen saturation by probe 56% and respiratory rate 41 cpm. On auscultation of chest bilateral diffuse inspiratory basal crepitations were found, more on the left side than right side. Jugular venous pressure of the patient was raised.

An immediate Arterial blood gas revealed severe hypoxia and increased lactate (Fig.1).

On further investigations serum cardiac enzymes and total leukocyte count were grossly raised and the level of CPK MB was 25 U/l and that of Troponin T was 0.36ng/ml. Chest X-ray revealed features suggestive of asymmetric pulmonary edema (Fig. 2).

Electrocardiograph revealed sinus tachycardia with secondary ST-T changes (Fig. 3). Echocardiogram

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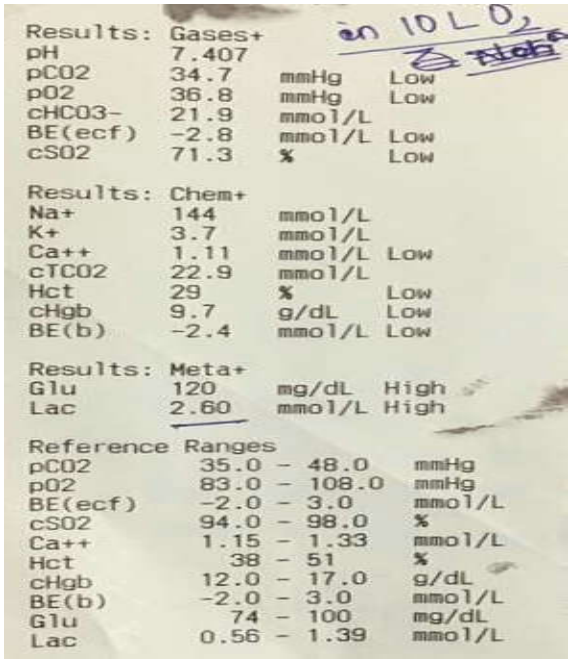
demonstrated dilatation of all 4 chambers with hypokinesia of interventricular septum and inferior posterior wall, moderate MR and TR with severe Left ventricular dysfunction (LVEF 23%). She was initially started with oxygen through a high flow oxygen mask but due to persisting low saturation level patient was put on non-invasive ventilation with high PEEP. Intravenous fluids could not be given as patient was assumed to be in fluid overload status. Inotropes noradrenaline and dobutamine were started along with diuretics infusion at a slower rate.

Total fluid intake of the patient was restricted. Her admission course was smooth and she was weaned of non invasive ventilation on the second day of admission and weaned of inotropes on the 3rd day of admission. She was shifted to ward on the 4th day and subsequent xray showed resolution of pulmonary edema and echocardiogram showed improved left ventricular ejection fraction. She was discharged on the 5th day of admission and is due for follow up one month later.



Results: Gases+			
pH	7.407		
pCO2	34.7	mmHg	Low
pO2	36.8	mmHg	Low
cHCO3-	21.9	mmol/L	
BE(ecf)	-2.8	mmol/L	Low
cSO2	71.3	%	Low
Results: Chem+			
Na+	144	mmol/L	
K+	3.7	mmol/L	
Ca++	1.11	mmol/L	Low
cTCO2	22.9	mmol/L	
Hct	29	%	Low
cHgb	9.7	g/dL	Low
BE(b)	-2.4	mmol/L	Low
Results: Meta+			
Glu	120	mg/dL	High
Lac	2.60	mmol/L	High

Reference Ranges		
pCO2	35.0 - 48.0	mmHg
pO2	83.0 - 108.0	mmHg
BE(ecf)	-2.0 - 3.0	mmol/L
cSO2	94.0 - 98.0	%
Ca++	1.15 - 1.33	mmol/L
Hct	38 - 51	%
cHgb	12.0 - 17.0	g/dL
BE(b)	-2.0 - 3.0	mmol/L
Glu	74 - 100	mg/dL
Lac	0.56 - 1.39	mmol/L

Fig. 1: Arterial blood gas showing severe hypoxia with increased lactate

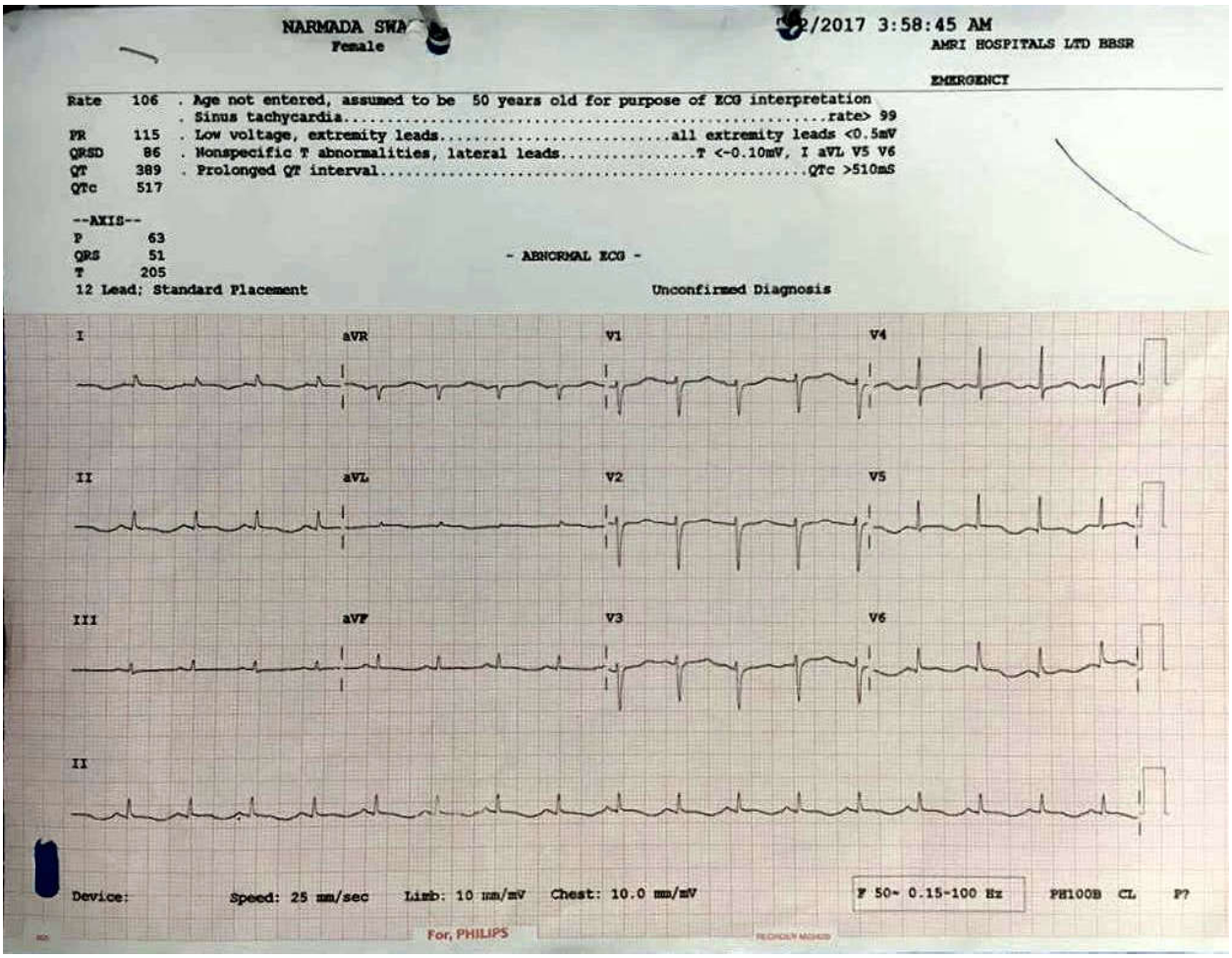


Fig. 2: Electrocardiograph showing sinus tachycardia with T inversions in inferior and lateral leads(I, II, II I, aVL , aVF, V4, V5, V6)

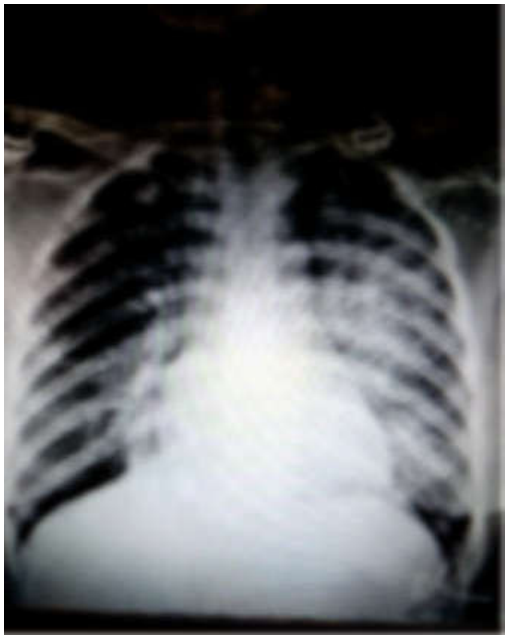


Fig. 3: Chest X-ray showing diffuse opacities in bilateral lung fields (left more than right) suggestive of asymmetric pulmonary edema

Discussion

The scorpion venom is a water soluble antigenic complex mixture of neurotoxin, cardiac-toxin, nephrotoxin, haemolysin, phosphodiesterases, phospholipase, hyalurinodases, histamine and other chemicals.These toxins are responsible for intense and persistent depolarization of autonomic nerves with massive release of endogenous catecholamines,an autonomic storm. 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].

Scorpion venom can cause myocardial damage by realising vasoactive, inflammatory and thrombogenic peptides and amine constituents (histamine, serotonin, bradykin in, leukotriens).

Which acts on the coronary vasculature and induce coronary artery vasospasm and facilitate platelet aggregation as well as thrombosis [6].

Direct cardiotoxic effect of the venom causes toxic myocarditis by reduction of Na-K+ At Pase and adrenergic myocarditis 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 [7].

Release of allergenic proteins causes anaphylactic shock leading to hypotension with vasodilatation and decreased intravascular volume with reduced myocardial perfusion [8].

Scorpion venom inhibits angiotensin converting enzyme (ACE) resulting in accumulation of bradykinin which is implicated in the development of pulmonary oedema.

Conclusion

Pulmonary edema is a common manifestation in scorpion bite but asymmetric pulmonary edema is quite rare. The mechanism of pulmonary edema induced by scorpion bite, though not completely understood, could be due to cardiogenic or non cardiogenic causes. The occurrence of pulmonary edema in our patient could be due to the reasons mentioned in discussion or severe left ventricular dysfunction as evidenced by Doppler echocardiography. What was striking was that pulmonary edema was asymmetrical and it developed more than 24 hours after the scorpion sting despite the use of prazosin early in the course.Patient also had hypotension, tachycardia and warm extremities. Though warm extremities could be due to prazosin therapy, the possibility of warm shock cannot be ruled out especially when the patient had breathlessness, tachycardia and hypotension.

Not only does this case emphasize the occurrence of asymmetrical pulmonary edema in scorpion sting,it also emphasizes that pulmonary oedema can occur late after sting and close monitoring of patients of scorpion sting is required beyond 24 hours as well by the Emergency physician and Critical care doctors.

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