

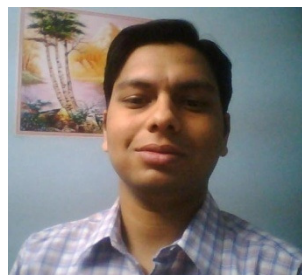
Scorpion Sting Envenomation With Pulmonary Odema & Hypotension: Treated With NTG & Inotropic Support: A Case Report

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Abstract

Scorpion sting is a major health problem in underdeveloped countries like India. It may present with mild local pain to severe systemic symptoms and need of I.C.U. care and mechanical ventilation. Scorpion envenomation presents with increased systemic vascular resistance and hypertension, but in cases of severe envenomation it may present with hypotension and pulmonary oedema. Here we are presenting a case of severe scorpion sting envenomation presented with hypotension and pulmonary oedema, which was treated successfully with NTG along with inotropes and mechanical ventilation.

Introduction

Scorpion Sting is a major health problem in many underdeveloped tropical countries. For every person killed by a poisonous snake, 10 are killed by poisonous scorpion¹. Out of 1500 scorpion species, 50 are dangerous to humans & can cause wide range of conditions from severe local reactions to neurologic, respiratory and cardiovascular collapse.

Scorpions are common in villages of TARAI & eastern area of Uttar Pradesh, India. The Indian Red scorpion (*Mesobuthus tamulus*) is common in these area and is one of the most toxic envenomation in animal kingdom.

Scorpion envenomation in humans is manifested as hypertension in mild cases and hypotension or pulmonary oedema in severe cases. Most

of the deaths due to scorpion sting are attributed cardiopulmonary complications like myocarditis and acute pulmonary oedema². As there are very few published studies of haemodynamic changes following scorpion envenomation, treatment of these patients has been essentially empirical or based on observations from animal studies³. The treatment of patients with hypotension and pulmonary oedema in particular is not clear⁴.

In India scorpion sting is very common in villages. Most of the cases are managed by local physicians at primary health centre and in district hospitals. Only cases with systemic manifestations are referred to higher centre. We received 21 cases in last one season. All of them presented with hypertension and increased SVR except 3, which presented with hypotension.

Here we are presenting a case report of scorpion sting envenomation admitted in our ICU with hypotension and pulmonary oedema.

Case Report

A 19 year female (Hospital No.19377) suffered with scorpion sting envenomation and came to us after 10 hours, having complaints of local pain at site of sting (right ankle), few episodes of vomiting and respiratory distress. Her heart rate was 120/min, BP 90/70 mmHg, SaO₂ 45%, having bilateral crepts in chest and frothy sputum coming from mouth. Her ABG was: pH:7.25, PCO₂:41.8, PO₂:32.6, HCO₃:18.2, BE:8.5, Na⁺:145, K⁺:4.9, Ca⁺⁺:1.29

The patient was intubated & put on Engstrom Ventilator SIMV mode with: Vt:350 ml, FiO₂:80%, RR: 10/min, PEEP: 8 cm of water, PS:14 cm of water.

The patient was treated with Inj. Amoxicillin+clavulanic acid 1.2gm iv tds, Inj. Metronidazole 100ml iv tds, Inj. Prazosin 0.5 mg iv stat followed by 0.5mg iv bd , Inj. NTG infusion @0.5 mic/kg/min. Inj. Dobutamine @ 10 mic/kg/min. Inj. Tetanus toxoid stat and Inj. Rabeprazole 20mg iv od. HR, NIBP, ECG, SpO₂, EtCO₂ & CVP were monitored continuously. Central venous pressure was on lower side. Blood sugar was monitored 2 hourly.

The patient was ventilated on SIMV mode over 12 hours, at that time her ABG was pH: 7.37, PaO₂: 85.0, PCO₂:37.3, HCO₃: 21.3, BE: - 3.5, Na⁺:148, K⁺:3.67, Ca⁺⁺: 0.989 At this time, NTG infusion was

stopped as her NIBP was going down. Dobutamine infusion was continued. Inj. Midazolam infusion @ 1mg/hr started to keep the patient sedated & for better tube tolerance.

After 36 hour the patient was shifted to CPAP mode with settings: FiO₂: 40%, PEEP: 5, PS: 10. Chest became clear. After 48 hour patient was given T piece trial and after successful trial she was extubated and maintained on ventimask with FGF: 5 lit/min. Incentive Spirometry was started. Dobutamine infusion was stopped, as she was maintaining normal BP. Her HR was 80/min, NIBP: 110/72 mmhg, SpO₂: 97%, CVP: 9 cm of water and the chest was clear. She was fully oriented and conscious. ABG was normal. Patient was monitored for 24 hours after extubation and discharged from ICU.

Discussion

Scorpion venom is a powerful stimulant of autonomic nervous system. The primary action of venom is through both sympathetic and parasympathetic postganglionic stimulation. In most of cases the sympathetic response predominates, resulting in "sympathetic storm"⁵ or "Autonomic storm"⁶. There is also a direct stimulant effect on the heart. The clinical presentation of scorpion sting covers a broad spectrum, known as "scorpion sting syndrome". Neale⁷ defined it as "varied manifestations of presumed scorpion envenomation." The typical can be described as local pain, occasionally with proximal radiation, often with tenderness, swelling and redness, followed by systemic symptoms which most commonly include hypertension and/or tachycardia, often with anxiety, nausea and epigastric pain. Cardiovascular manifestations are due to direct effect of excess circulating catecholamines and cholenergics from autonomic hyper stimulation. The sympathetic system of the autonomic nervous system usually predominates, resulting in hypertension and tachycardia and in case of severe envenomation, dysrhythmias, left ventricular failure and pulmonary oedema. Parasympathetic predominance may result in bradycardia, various grades of AV blocks, and non-cardiovascular manifestations such as priapism and hypersalivation.

Our patient presented in state of cardiogenic shock (tachycardia, hypotension, acidosis), respiratory distress, hypoxemia (SPO₂:45%, PaO₂: 32.6%) and pulmonary oedema. Pulmonary oedema after scorpion sting occurs only when the left ventricular end diastolic pressure is high though CVP remains low. Low central venous pressure indicates subnormal right ventricular preload and may not reflect left ventricular diastolic pressure

in acute left ventricular dysfunction⁴. Pulmonary wedge pressure was not measured. Late onset pulmonary oedema is due to acute myocardial injury and LVF caused by the toxin induced autonomic storm. This has been reported in 17%-34.8% cases from Saudi Arabia and India⁸

Factors like hypoxemia and hypercarbia contribute to pulmonary hypertension. Hyperoxygenation by positive pressure ventilation at high FiO₂ given to reduce pulmonary hypertension & to resolve pulmonary oedema.⁹ PEEP helped by alveolar recruitment and by shifting oedema fluid away from alveoli.

Haemodynamic control with adequate fluid replacement and inotropic support (dobutamine) treated hypotension and improved cardiac function. Dopamine is not used because it further increase the catecholamine induced cardiac damage.¹⁰ In severe cases dobutamine infusion is reported to improve impaired heart function¹¹

Usefulness of Prazosin in preventing cardiopulmonary complication had been described in adults. Prazosin and NTG infusion with CVP guided fluid was given to reduce afterload and better cardiac output alongwith dobutamine infusion as recommended by Niranjan Biswal et al¹². Bawaskar & Bawaskar (2000) also recommended prazosin medication to prevent & treat pulmonary oedema, as prazosin (a post synaptic alpha-1 blocker) has the pharmacological properties that counteract the effects of excessive catecholamines & help in reducing pulmonary congestion and has been found an effective drug for scorpion sting¹³.

Antivenom therapy was not used because it is species specific and works only when it is given immediately after the sting¹⁴. Besides, recent reports have disfavoured its use in preventing cardiovascular manifestations for Indian species of scorpion venom¹⁵.

Delayed hospitalization was associated with severe life threatening complications. This report emphasizes the complexity of clinical picture and need of intensive approach to timely diagnosis of pulmonary oedema and initiation of NTG with ventilatory and inotropic support in ICU for better outcome.

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