

A Case Series of Indian Red Scorpion Bite and Rare Clinical Presentation of Central Nervous System

Dr. Nirav Patel¹, Dr. Megh Shah², Dr. Yash Patel³

¹ Assistant professor, Department of Medicine, B. J. Medical College and Civil Hospital, Ahmedabad

^{2,3} IInd year resident, Department of Medicine, B. J. Medical College and Civil Hospital, Ahmedabad.

Corresponding Author: Dr. Nirav Patel

Email: nirav25nhl@gmail.com



Abstract

Indian red scorpion (*Hottentotta tamulus*) is a very dangerous venomous animal found in Indian Territory; its venom contains various toxins which causes direct effect on the central nervous system (CNS). We report a case series of 12 patients who were bitten and had atypical clinical presentation affecting CNS. We evaluated 12 patients of Indian red scorpion sting by taking history, routine blood investigations, CT scan of brain, and CNS examination at a tertiary care hospital in Gujarat. We observed that if patient takes longer time after sting to visit hospital, there are more chances of development of CNS signs and symptoms and indicates poor prognosis. Indian red scorpion venom is a potent sympathetic stimulant and cardio-toxic agent. It can also cause haemorrhagic and thrombotic stroke, cerebellar stroke and cerebellitis. Prazosin is the treatment of sting as there is no availability of anti-venom for sting. Prognosis is directly related to duration between scorpion sting and initiation of treatment.

Keywords: 5' nucleotidase, roving eye movements, acute cerebellitis.

Introduction

The Indian red scorpion (*Hottentotta tamulus*), also known as the eastern Indian scorpion, is a species of scorpion of the family Buthidae, order: scorpions, phylum: arthropoda, subphylum: chelicerata and belongs to animalia kingdom. Indian red scorpion belongs to arachnid class of animal. It occurs in most of India, eastern Pakistan and the eastern lowlands of Nepal. It bites humans more in night time than daytime commonly during months of April to August. Scorpion venom is water soluble, antigenic, heterogenous mixture. This heterogeneity accounts for the variable patient reaction to Indian red scorpion sting. Indian red scorpion venom contains toxins like polypeptides and various enzymes like acetyl cholinesterase, alkaline phosphatase, acid phosphatase, 5' nucleotidase, hyaluronidase, ribonuclease and deoxy-ribonuclease.¹ This water soluble mixture of toxins and enzymes exerts toxic effect on neurons and is responsible for rare clinical features like roving eye movements and increased tendon reflexes with prolongation of relaxation phase.

Case series

We report a case series of 12 patients admitted in Department of Medicine at a tertiary care hospital in Gujarat, who were bitten by Indian red scorpion and developed rare CNS manifestations. Most patients presented within 3 days of sting. Most common among rare features observed was roving eye movements. Out of 12 patients, 11 (91.6%) had roving eye or rotatory eye movement. All patients complained of intense pain and paraesthesia. Twitching and piloerection accompanied by goose pimples were also present in 3 (25%) patients. Five (41.6%) patients had involuntary muscle spasm and alternating opisthotonos. Table 1 describes clinical features observed in enrolled patients following sting bite.

Table 1: Details of patients presenting with Indian red scorpion sting, clinical features and treatment given

Features	Case											
	1	2	3	4	5	6	7	8	9	10	11	12
Age (Years)	34	60	18	22	27	53	74	60	57	44	19	23
Sex(M/F)	M	M	F	F	M	M	M	F	M	M	F	F
Sting to Hospital Time (In days)	2	4	3	3	5	1	3	3	5	3	3	5
Vomiting	+	++	+	+	++	-	-	+	+++	+	-	+
Altered Sensorium	No	No	No	No	Yes	No	No	No	Yes	No	No	Yes
Pink Frothy Sputum	-	+	+	+	++	-	-	-	+	+	-	+
Temp (⁰ F)	99	98	100	97	99	98	97	99	101	99	96	100
Pulse (bpm)	88	94	112	80	56	74	100	88	114	82	84	72
Blood Pressure (mm Hg)	130 /84	120 /80	110 /74	150 /90	180 /98	130 /84	140 /90	124 /70	200 /96	160 /88	136 /78	146 /90
Roving Eye Movement	+	-	+	+	+	+	+	+	+	+	+	+
Clonus	+	+	+	-	+	-	+	+	+	+	+	+
Increased DTR and Delayed Relaxation	+	+	+	-	+	-	+	+	+	+	+	+
Dysmetria	-	+	-	-	-	-	-	-	+	-	-	-
Broad Based Gait	-	-	-	-	-	-	-	+	-	-	-	+
Stroke(H/I)	-	-	-	-	+(H)	-	-	-	+(H)	-	-	-
Prazosin& Supportive Treatment given	Yes											
Survived	Yes	No	Yes	Yes	No	Yes	No	Yes	No	Yes	Yes	No
Ramipril (ACEI)given	No	No	No	Yes	Yes	No	Yes	No	Yes	Yes	No	No

*H=hemorrhagic stroke *DTR= deep tendon reflexes)

Other common sympathetic features like hyperthermia, tachycardia, hypertension and pulmonary edema and parasympathetic features like bradycardia, hypotension, miosis and priapism were also present in all 12 (100%) patients. All patients presenting to us with rare CNS manifestations were not treated in the early days. As history given by the patients or patients' relatives, symptoms like shortness of breath, fever, painful erection of penis occurred within a few minutes after the sting and usually progressed to severe form within 7 to 8 hours. We observed that out of 12 patients, ten (83%) developed UMN signs and five (41.6%) of these died (four patients died due to encephalopathy and one patient died because of cardiac complications). Of them, two (16.6%) patients had stroke and raised intra cranial tension. Patients with dysmetria and broad-based gate also had intention tremor, nystagmus, ataxia, scanning speech and hypotonia. Patients with cerebellitis with cerebellar signs gradually improved with steroids. Four (33.33%) patients suffering from dilated cardiomyopathy were treated with prazosin and ACE inhibitors and one (8.33%) patient developing rhabdomyolysis was treated with hemodialysis. Of them, 2 (40%) patient died because of stroke, 1 (20%) patient died because of cardiac complications while 2 (40%) patients improved with ACE inhibitors. A total of 11 (91.6%) patients developed persistent paraesthesia, and four (33.33%) patients developed ankylosis of small joints and were treated accordingly.

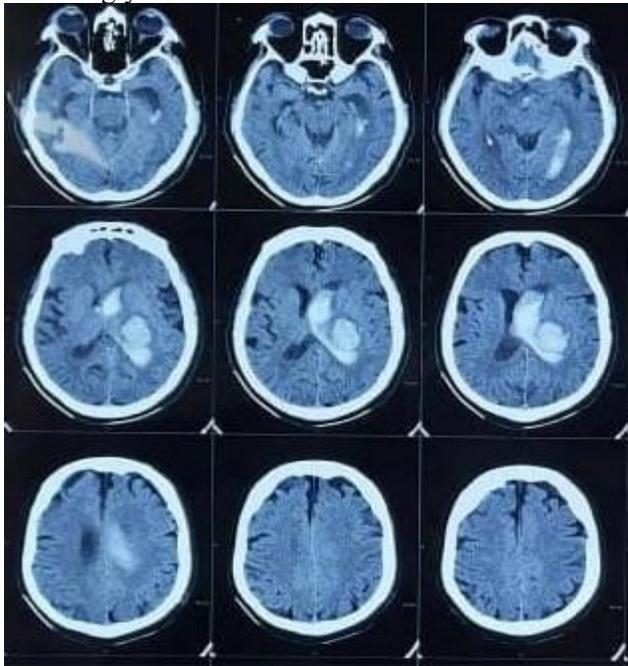


Fig-1 Findings of Haemorrhagic stroke in CT scan (without contrast) of a 27 years old male suffering from Indian red scorpion sting

Discussion

The signs of the envenomation are determined by the scorpion type, venom content and the patient's physiological response to the venom. 5' nucleotidase and acid phosphatase of venom is responsible for the roving eye movement.² Most roving eye movements are predominantly horizontal although vertical movements also occur. These movements are because of cortical dysfunction. Water soluble content of venom also exerts a toxic effect on neurons and causes rhythmic, shaking movements and clonus. We observed 83% patients with clonus and contractures following sting bite (table -1). The signs of scorpion bite last for 2 – 5 days and do not have an apparent sequence. So prediction of development of signs overtime is difficult.³ We also reported 83% patients with increased deep tendon reflexes with

prolongation of relaxation phase. Venom's water soluble contents like 5' nucleotidase and deoxyribonuclease exerts direct toxic injury or produces catecholamine storm and produces upper motor neuron signs.⁴ As a result, patient develops exaggerated reflexes with increased relaxation, spasticity and rigidity like somatic effects. As in sting patient develops sympathetic and parasympathetic symptoms both alternatively, we also found same in all of our patients.

Out of 12 patients, five (41.6%) patients died. We reported 2 cases of hemorrhagic stroke (fig-1) Scorpion venom can lead to either hemorrhagic or ischemic stroke. This can be because of contents of venom like alkaline phosphatase, hyaluronidase and acid phosphatase or complications such as anaphylactic shock or pain produced by catecholamine storm. We also reported 33.33% patients developing cerebellar signs like dysmetria (16.66% patients) and broad based gait (16.66%patients). Indian red scorpion venom is very toxic and sometime may produce infarct in cerebellum by its toxic content like polypeptides and cause cerebellar dysfunction. Sometimes venom causes inflammation of the cerebellum.⁵ Patients with cerebellar signs also had ataxia and hypotonia which is very rare in sting bite.⁶ A false recovery followed by a total relapse is common.

Treatment with prazosin – a competitive postsynaptic alpha 1, adreno-receptor antagonist is first line treatment of scorpion bite. It suppresses sympathetic outflow, activates potassium channels, decreases preload, afterload and blood pressure without increasing heart rate. It may also prevent CNS manifestation and improve the prognosis. Delay in treatment with alpha blocker may lead to development of rare clinical CNS manifestations. Recommended dose is 30 microgram/kg/dose. Venoms reach their target tissues too rapidly, so anti-venom is required within 30 mins to reverse effect, however anti-venom against Indian red scorpion is not available.⁷

Cardiac manifestations like dilated cardiomyopathy, coronary vasospasm and myocardial ischemia are common in Indian red scorpion bite. Water soluble content of venom is cardiotoxic, which causes reduction of Na-K- ATPase and leads to myocarditis. It also leads to secretion of catecholamines from ganglia and adrenals and causes increase in demand of oxygen in myocardium. Sting venom also causes release of inflammatory peptides which is responsible for ischemia and coronary spasm.⁸ Angiotensin converting enzyme inhibitor improves the diuretic resistant pulmonary oedema and cardiogenic shock due to scorpion sting.⁹ So, Five patients who developed hypertension with shortness of breath and signs of raised intra cranial tension were treated with ramipril. Of them, 2 (40%) patient died because of stroke and 1 (20%) patient died because of cardiac complications while 2 (40%) patients improved with ACE inhibitors. Other Patients with roving eye movements and UMN signs gradually recovered completely with symptomatic treatment. Patients with cerebellar infarct also improved after treatment with anti-platelets and physiotherapy.

Conclusion

Indian red scorpion venom is a potent sympathetic stimulator and cardiac manifestations are common with Indian red scorpion sting. CNS involvement indicates poor prognosis. Cerebellitis and cerebellar infarct are rare presentations with Indian red scorpion. Cerebellitis can be treated with steroids. Duration of time between sting and administration of prazosin, decides the prognosis.

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