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CASE REPORT

Demyelinating Polyneuropathy Following Scorpion Sting Envenomation; a Case Report and Review of Literature

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Abstract

Background: Scorpion sting envenomation generally causes treatable local and systemic effects; however, in rare cases, the victims might experience sequels in end organs such as central nervous system. In the present paper, a case of relatively self-limiting demyelinating polyneuropathy following a Butidae sting is presented and the possible mechanisms are discussed.
Case Presentation: A 19-year-old man presented to emergency department of Sultan Qaboos University Hospital, Oman with severe throbbing pain at the base of his right big toe after a scorpion sting. His initial examination revealed normal vital signs and the systemic examinations were unremarkable. Few minutes later, he developed profuse sweating, slurred speech, blurred vision, increased salivation and restlessness. Repetition of measurement of vital signs showed a blood pressure of 160/100 mmHg, heart rate of 140 beat per minute and a respiratory rate of 18 per minute. The patients received scorpion antivenom and cholinergic hyperactivity manifestations. Shortly after, the patient developed involuntary jerky movements in both lower associated with fasciculation. Nerve conduction study was suggestive of demyelinating polyneuropathy. In later days, involuntary jerky movements of lower limbs improved gradually but fasciculation remained. On a follow-up visit after four months, the patient still complained of occasional fasciculation.
Discussion: One explanation for the development of peripheral nerves demyelination in our patient is the inflammatory response triggered by scorpion venom. In addition, this complication can be attributed to direct cytotoxic effects of scorpion venom toxins. Antimicrobial peptides in scorpion venoms are shown to be highly toxic to human cells, which in our case might have damaged the nerve sheet.
Conclusion: Severe scorpion sting envenomation may lead to severe systemic effects and end organ damage. Medical toxicologists should be prepared to diagnose and treat such sequels.

Keywords: Fasciculation; Myoclonus; Oman; Polyneuropathies; Scorpion Stings

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INTRODUCTION

Scorpion sting is a common health problem in tropical areas and countries with desert biomes (1,2). In Oman, scorpion stings accounts for nearly one-fifth of human casualties due to animal bites and stings (3). Native venomous scorpions in Arabian Peninsula commonly belong to Buthidae family and include genera of Buthacus, Leiurus, Androctonus, and Vachoniolus (4-6). The venom of these scorpions contains neurotoxins that trigger endogenous acetylcholine and catecholamine release (1). Hence, a set of manifestations similar to the cholinergic syndrome of organophosphate poisoning will be developed (1,7).

In the majority of cases, scorpion sting envenomation causes treatable local and systemic effects. However, in some cases, the victims might experience sequels in end organs such as kidneys, heart, and peripheral and central nervous system (1,8). In the present paper, a case of relatively self-limiting demyelinating polyneuropathy following a Butidae sting in Oman is presented and the possible mechanisms are discussed.

CASE PRESENTATION

A 19-year-old man presented to emergency department (ED) of Sultan Qaboos University Hospital with severe throbbing pain at the base of his right big toe after a scorpion sting. The scorpion subsequently identified as belonging to the Buthidae family. His initial examination revealed minimal redness and tenderness at the base of his right big toe, while vital signs and the systemic examinations were unremarkable.

Few minutes later, he developed profuse sweating, slurred speech, blurred vision, increased salivation and restlessness. Repetition of measurement of vital signs showed a blood pressure of 160/100 mmHg, heart rate of 140 beats per minute

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Table 1. Laboratory values of the patient in two time-points (baseline at emergency department and during admission to hospital ward)

Parameter	Unit	Normal range	Baseline*	Second measurement**
Hemoglobin	g/dL	11.5-15.5	16.9	15.7
WBC count	× 10^3 cells/mm^3	2.2-10.0	9.4	4.8
Hematocrit	%	35-45	49	45
Platelet count	× 10^3 cells/mm^3	140-400	210	181
Serum Na	mEq/dL	135-145	143	135
Serum K	mEq/dL	3.5-5.1	4.0	4.4
Creatinine	µmol/L	59-104	77	69
Urea	mmol/L	2.8-8.1	4.2	6.5
ALT	U/L	0-40	25	22
AST	U/L	0-40	28	24
Lipase	U/L	13-60	19	NA
Troponin I	µg/L	< 0.04	<0.01	NA
CK-MB	U/L	0-25	21.7	NA
Lactate	mEq/L	0.5-2.2	3.1	NA
PT	Sec	10-11.8	11.4	12.5
aPTT	Sec	28.2-39.8	32.8	38.3
INR	-	0.9-1.05	1.02	1.11

WBC: White Blood Cell; ALT: Alanine Aminotransferase; AST: Aspartate Aminotransferase; CK: Creatine Kinase; PT: Prothrombin Time; aPTT: Activated Partial Thromboplastin Time; INR: International Normalized Ratio
NA: Not assessed
* At emergency department
** During admission to medical ward

and a respiratory rate of 18 breaths per minute. His oxygen saturation was 100%. There was no nystagmus, fasciculation or jerky movements. On electrocardiogram, sinus tachycardia was evident. As shown in table 1, except a mild rise in baseline lactate level, all laboratory investigations of the patient were within normal limits both in baseline at ED, and later during admission to medical ward.

After supportive treatments, the patient received 3 vials of polyvalent scorpion antivenom (National Antivenom and Vaccine Production Center, Riyadh, KSA). Shortly after, cholinergic hyperactivity signs and symptoms subsided dramatically. Three hours later, he started to develop similar manifestations which required additional 2 vials of the antivenom. Almost seven hours after admission, the patient developed involuntary jerky movements in both lower limbs but more often in left side associated with fasciculation. Hence, nerve conduction study (NCS) was requested for the patient. In NCS, reduction in motor conduction velocity, prolonged minimum F wave latencies, decreased sensory nerve conduction velocity, and reduced CMAP and SNAP amplitudes were detected. These findings were suggestive of demyelinating polyneuropathy. Hence, moderate dose benzodiazepine was started for the patient to control myoclonus and fasciculation. In later days, involuntary jerky movements of lower limbs improved gradually but fasciculation remained. With advice of a close follow-up visit, the patient was discharged on clonazepam, three days post- admission. On a follow up visit after four months, the patient still complained of occasional fasciculation.

DISCUSSION

Butidae scorpion stings are generally associated with severe local effects and sometimes systemic effects that are favorably responsive to supportive treatments and scorpion specific antivenom (1). Nonetheless, a small number of victims might be affected by prolonged idiopathic complications, which in our case it showed itself with long-lasting polyneuropathy. To the best of our knowledge and according to broad-range literature review, this was the first report of demyelinating polyneuropathy following scorpion sting envenomation. Butidae scorpions (Figure 1) are notorious for the neurotoxic components in their venoms (9). However, these neurotoxins hardly leave sustained tissue damages if the patient is treated early with a right amount of antivenom. Scorpion neurotoxins can block the function of specific ion channels in excitable cells resulting in autonomic excitation. These toxins can be classified into two main types, namely α-toxins and β toxins. The α-toxins can induce massive release of catecholamines and consequently cholinergic hyperactivity (9). However, this set of events may not justify the demyelination of peripheral nerves.

One explanation for the development of peripheral nerve demyelination in our patient is the inflammatory response triggered by scorpion venom. Following severe scorpion envenomation, high levels of pro-inflammatory cytokines occurs that contribute to immunological imbalance and

Figure 1. A Butidae scorpion (*Hottentotta jayakari*) similar to the scorpion that offended our patient (6)

tissue/organ damage (9,10). In addition to this, the occurs that contribute to immunological imbalance and tissue/organ damage (9,10). In addition to this, the complication of our patient can be attributed to direct cytotoxic effects of scorpion venom toxins. Antimicrobial peptides in scorpion venoms are shown to be highly toxic to human cells which in our case might have damaged the nerve sheet (9,11). Nonetheless, nitric oxide (NO) production might be the co-etiology of consciousness impairment in scorpion sting envenomation, as it can cause vasodilation and reduced cranial circulation during acute inflammation (10). It has been advocated that cytokine-mediated circulatory shock is caused by activation of the inducible isoform of NO synthase (10,12).

In handful of papers, severe neurologic complications following scorpion stings are reported. One article presented a patient who suffered dysarthria and flaccid quadriparesis after being stung by an unknown kind of scorpion and completely recovered after antivenom administration (13). A retrospective study, which included 111 Egyptian children with scorpion sting, revealed that 53.2% of them had severe envenomation and 22.8% of them developed severe neurological manifestations including convulsions and coma (14). Correspondingly, Uluğ et al reported a child with seizure after scorpion sting in Turkey (15). Scorpion envenomation is also found to be associated with multiple intracranial parenchymal hemorrhages in frontoparietal and high parietal area (16). In another paper, Cavari et al reported severe envenomation by *Leiurus quinquestriatus hebraeus* causing multi-organ failure manifested with seizure, coma, severe brain edema, non-cardiogenic shock, disseminated intravascular coagulation, renal failure and hepatic failure in a two-year old boy, which finally led to death (17). They concluded that direct CNS toxicity (penetration of the toxins through the blood-brain barrier, affecting the CNS neurons) or unusual secondary effect of neurotransmitters and pro-inflammatory cytokines on blood vessels were the possible underlying mechanisms of brain ischemia and cytolytic brain edema (17). A prospective study on 50 Indian patients with scorpion sting showed 8% of them had cerebrovascular accident; half of them had hemorrhagic stroke and another half developed thrombotic stroke (18). In another case series, Bosnak et al reported 19% of 52 pediatric victims stung by *Androctonus crassicauda* or *Leiurus quinquestriatus* had impaired consciousness while subsequently half of them developed deep coma probably due to hypertensive encephalopathy or brain ischemia (19). Interestingly, 3 of the reported patients finally developed persistent muscle cramps (19), an event that resembles residual lower limb fasciculation of our patient.

The antivenom available in Oman, which is produced by the national Saudi antivenom production center, is a $F(ab)^2$ equine derived polyvalent scorpion antivenom. It is highly specific in neutralizing the venoms of death stalker yellow scorpions (*Leiurus quinquestriatus*), Arabian fat-tailed black scorpion (*Androctonus crassicauda*) and variety of other scorpions native to Arabia. It can also neutralize the venoms of many of Middle East and North African scorpions including *Buthus arenicola, Butus mimax, Buthus occitanus, Leiurus quinquestriatus hebreus and Androctonus amoreuxi* (20).

LIMITATIONS

The puncture mark of the scorpion sting was evident on our patient, and we based our diagnosis on clinical findings and patient's history; however, we could not be sure about the offending species, as no toxicological analysis for venomous creatures is available in our setting.

CONCLUSION

Severe scorpion sting envenomation may lead to severe systemic effects and end organ damage. Emergency physician should be prepared to diagnose and treat such sequels. The antivenom therapy is vital and effective to reverse the scorpion venom induced effects. It should be available in all hospitals in the vulnerable regions.

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