

CARDIAC ARREST FOLLOWING SCORPION ENVENOMATION

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Scorpion stings are a common occurrence in Saudi Arabia. The majority of cases present with local pain at the site of envenomation, and follow a benign clinical course. Severe systemic toxicity and mortality are uncommon. Reported deaths have been attributed to a primary effect of the venom on the cardiovascular system. Toxicity of envenomation varies between species and within the same species of scorpions in different geographic regions. Severe systemic toxicity is rarely encountered in the Central Province of Saudi Arabia.¹ The case presented is that of cardiac arrest, following scorpion envenomation in a 51-year-old male from the Riyadh area. This complication of envenomation is highly unusual. Mechanisms of venom toxicity and management guidelines are discussed.

Case Report

A 51-year-old Saudi male presented to the Emergency department 20 minutes after envenomation in the left palm by a "large black scorpion" (*Androctonus* spp). The patient complained of severe local pain radiating proximally up the left arm. He was noted to be pale and diaphoretic, with a blood pressure of 177/90 mm Hg, pulse of 104, respiratory rate of 30 breaths per minute and temperature of 37.2°C. The patient was a non-smoker, with a history of non-insulin dependent diabetes mellitus. There was no history of hypertension or cardiac disease.

Twenty-five minutes after arrival, the patient suddenly collapsed. He was found to be in full cardiopulmonary arrest with ventricular fibrillation. The patient was resuscitated as per Advanced Cardiac Life Support protocol. Repeated defibrillation attempts, intravenous epinephrine and lidocaine were given. Five ampoules of scorpion antivenin were also infused during the resuscitation. After 30 minutes, the patient was finally converted to a perfusing sinus rhythm. A follow-up

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electrocardiogram (EKG) revealed a 2 mm ST-segment elevation in leads II, III and AVF, suggesting an inferior wall myocardial infarction. The patient was transferred to the Intensive Care Unit (ICU) for further management. Physical examination at the ICU showed bilateral fixed and dilated pupils. Vasopressor therapy was initiated for refractory hypotension. Thirty minutes into the ICU stay, the patient again had an episode of unstable ventricular tachycardia, which responded to intravenous amiodarone and a continuous amiodarone infusion. A bedside transthoracic echocardiogram showed acute inferior wall motion abnormalities with global hypokinesia and an estimated left ventricular ejection fraction of 35%. Post-arrest laboratory data showed acute hepatic and renal failure that were felt to be a consequence of his prolonged cardiac arrest. The patient's creatinine phosphokinase level rose to 14,300 IU, with 33% CKMB fraction.

The patient completed 5 doses of antivenin for the scorpion sting and aggressive supportive care was continued. The next 24 hours failed to show any sign of neurological improvement. CT scan of the head showed diffuse cerebral edema consistent with severe anoxic insult. EEG on the third day of ICU stay was iso-electric. Physical examination showed an absence of brain stem function off sedation. Renal failure progressed, resulting in anuria, and hemodialysis was initiated. By the fifth day, a ventilator-associated pneumonia had developed and antibiotics were commenced. Unfortunately despite all efforts, the patient's condition continued to deteriorate and he expired on the 10th day of ICU care.

Discussion

Scorpions are widely distributed in Saudi Arabia. Fourteen species or subspecies belonging to two families, the *Buthidae* and the *Scorpionidae*, have been identified.² Nine of these have been found in the Riyadh area alone. The two most venomous species appear to be *Leiurus quinquestriatus* and *Androctonus crassicauda* of the family *Buthidae*. They are more commonly known as the "yellow scorpion" and "black scorpion," respectively.

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Scorpions are nocturnal. They are unable to tolerate the high temperatures of the day, and seek protection from the heat by sheltering under rocks or debris.^{3,4} Both their nocturnal predatory pattern and their seasonal hibernatory cycle, are reflected by the higher incidence of patient contacts during the evening and night-time hours of the summer months.

Scorpion venom is composed of a variety of substances including mucus, polynucleotides, small organic molecules, salts, and nontoxic proteins.⁵ The venom of some scorpions also contains a number of basic polypeptides and neurotoxins.^{6,7} Toxicity in scorpions is variable, even within the same species, and from place to place. It also varies with the size, age and nutrition of the scorpion as well as climatic conditions.⁸ The clinical effect of the venom is also affected by the body mass and the general health of the patient.^{7,9} The very young and elderly are most susceptible to developing systemic symptoms of envenomation. Diabetic and hypertensive patients are also at higher risk of developing systemic toxicity.¹

Scorpion venom is a powerful stimulant of the autonomic nervous system. The primary action of venom is through both sympathetic and parasympathetic post-ganglionic stimulation. In most circumstances, the sympathetic response predominates, resulting in what has been described as a "sympathetic storm."⁹ There is also a direct stimulant effect on the heart.¹⁰

The clinical presentation of scorpion stings covers a broad spectrum. The vast majority of patients experience no more than localized pain.¹¹⁻¹⁴ "Scorpion sting syndrome" was defined by Neale to be "the varied manifestations of presumed scorpion envenomation." The typical case can be described as local pain, occasionally with proximal radiation, often with tenderness, swelling and redness at the site of envenomation. This may be followed by the onset of systemic symptoms, which most commonly include hypertension and/or tachycardia, often with anxiety, nausea and epigastric discomfort.¹² With more severe envenomation, neuromuscular hyperactivity can occur. This may manifest itself as peripheral motor jerking, cranial nerve fasciculations or opisthoclonus. This is often misinterpreted as being a seizure.^{13,15} Metabolic acidosis and hyperthermia may result from this excessive muscular activity.¹³

In a prospective study by Soomro et al., it was found that all patients presenting with neurologic complications also had evidence of cardiovascular toxicity.¹⁶ Cardiovascular manifestations are due to the direct effects of excess circulating catecholamines and cholinergics from autonomic hyperstimulation. The sympathetic branch of the autonomic nervous system usually predominates, resulting in hypertension and tachycardia, and in cases of severe envenomation, dysrhythmias, left ventricular failure and pulmonary edema. Parasympathetic predominance may result in bradycardia, various grades of AV blocks, and non-cardiovascular manifestations such as priapism and

hypersalivation. The cardiovascular effects of severe toxicity are the primary cause of death.^{8,9,16-18} They can be

divided into five different syndromes: hypertension, pulmonary edema with hypotension, and rhythm disturbances.¹⁹

Localized or global ventricular hypokinesia with left ventricular systolic dysfunction appears to be the cause of pulmonary edema when it occurs. This effect on the myocardium appears to be the result of catecholamine excess, as well as direct myocardial damage by the venom.^{18,19} The most significant electrocardiographic changes caused by scorpion venom include myocardial ischemia, anterior or inferior myocardial infarction, and malignant dysrhythmias. Myocardial infarction has been confirmed to occur in the setting of severe envenomation, through enzyme elevation and post-mortem examination.¹⁹ EKG changes simulating myocardial injury or infarction, has also been described.¹⁶ Recovery of cardiac function, as determined by echocardiography, is often delayed by up to one month. Clinical recovery of cardiovascular function, however, usually occurs sooner, within 5 to 12 days.¹⁶

If severe envenomation is to occur, it usually appears within the first six hours following a sting.^{11,13,20}

Experimentally, autonomic stimulation causes significant biochemical disturbance, such as hyperglycemia, hypocalcemia, hypophosphatemia and elevation in serum alkaline phosphatase.^{21,22} However, with the exception of transient hypocalcemia, no significant hematological or biochemical findings are seen clinically.²³

The use of antivenin has been subject to a great deal of controversy. Its efficacy has recently been challenged, with questions raised as to its clinical benefit.^{15,24} In the only randomized, blinded, controlled trial assessing the efficacy of antivenom, no benefit was found, irrespective of clinical severity.²⁴ Other studies support its use citing a resolution of morbidity and a reduction in mortality.^{15,19,25,26} Antivenin still continues to be recommended in grade 3 and 4 envenomations (Table 1), and is said by some authors to be the treatment of choice for severe neuromuscular hyperactivity.^{1,13,15} It has been reported that antivenin will resolve symptoms within 1-3 hours of administration, and those patients not receiving antivenin, possibly having symptoms persisting for 9-30 hours.¹⁵ This has been questioned by some authors, arguing that there is no conclusive evidence that antivenin abolishes or reduces venom effect on the autonomic nervous system.²⁷

The majority of scorpion stings fortunately pursue a benign course, and can be safely treated with supportive measures alone, without the use of antivenin. The management of a scorpion sting is based upon initial categorization of the grade of envenomation (Table 1). Initial history and physical examination should help define the grade of envenomation and identify those in high-risk groups for serious toxicity. High-risk patients are those aged less than 10 years or greater than 50 years; history of

hypertension or diabetes; or patients who have sustained multiple stings.¹

TABLE 1. *Grades of envenomation.*

Grade	Description
I	Local pain and/or erythema and/or paresthesia at site of envenomation
II	Pain and/or paresthesia remote from the site of the sting and/or tachycardia and mild hypertension in addition to local findings
III	Cranial nerve or somatic skeletal neuromuscular dysfunction or cardiovascular dysfunction
	Cranial nerve dysfunction Blurred vision, wandering eye movement, hypersalivation, trouble swallowing, tongue fasciculation, problems with upper airway and slurred speech
	Somatic skeletal neuromuscular dysfunction Jerking of extremity, restlessness, severe involuntary shaking and jerking that may be mistaken for a central seizure disorder
	Cardiovascular dysfunction Moderate to severe hypertension, cardiac dysrhythmias, myocardial ischemia, pulmonary edema
IV	Any combination of cranial nerve, somatic skeletal neuromuscular or cardiovascular dysfunction

A system of defining the grade of envenomation (Table 1) assists in directing therapy of the patient.¹ With grade I and grade II envenomation, pain management is the mainstay of therapy. Depending on the patient's level of discomfort, local and/or systemic analgesia can be given. Local infiltration of the sting site with either lidocaine or bupivacaine, has been shown to be effective in many patients.¹ Application of ice to the site of envenomation often provides some relief of pain. Systemic analgesia, either oral or parenteral, may provide additive relief. Agents such as acetaminophen (+/- codeine), nonsteroidal anti-inflammatories, and narcotics are safe and effective in recommended dosages.¹ The large majority of patients will fall into this group and will require no further intervention.

The period of observation is dictated by clinical condition and response to pain management. Patients may be safely discharged after two hour if they do not fall into one of the high-risk categories and their vital signs remain within normal limits. For those patients who are in the high-risk group, further observation is recommended. Patients with tachycardia or mild hypertension should be observed until vital signs return to normal. Observation beyond six hours from the time of the sting does not appear to be justified unless serious toxicity occurs.^{13,15}

Grades III and IV envenomation require full supportive measures as necessary. Intravenous access and cardiac monitoring are required as well as supplemental oxygen if indicated. Antivenin should be given to these patients. Local and systemic analgesia, as described for grades I and

II envenomation, can be provided as necessary. All these patients should be admitted to the hospital and followed in an intensive care setting until their condition stabilizes.

Our patient presented early on with evidence of grade III envenomation. His condition rapidly deteriorated into full cardiopulmonary arrest. The development of ventricular fibrillation may have been due to a number of factors. It could possibly have resulted from a primary malignant rhythm disturbance, directly resulting from venom toxicity on the myocardium. Another possibility is that the "sympathetic storm" brought on by envenomation led to myocardial infarction followed by ventricular fibrillation. Because an electrocardiogram was not done prior to the patients collapse, it is impossible to know whether the EKG changes suggesting infarction were present prior the onset of his arrest. The development of ventricular fibrillation, especially so soon after envenomation (i.e., less than one hour) was highly unusual. This case helps to emphasize the need to assess patients with scorpion sting in a timely fashion, and be aware of the potential for life-threatening complications of envenomation.

Scorpion stings are common in the Middle East. The great majority of cases in otherwise healthy subjects can be conservatively treated by adequate analgesia, the local application of ice, and a period of observation dictated by the clinical findings. Particular care should be taken in the assessment of the very young and old, and to those with pre-existing morbidity, such as hypertension and diabetes. Serious envenomation is manifested primarily by cardiovascular toxicity. Excess sympathetic stimulation, as well as a direct effect of venom on the myocardium are responsible for the most serious cardiac manifestations of toxicity. Because scorpion stings are associated with known instances of serious morbidity and mortality, the place for antivenin in cases displaying serious (grades III and IV) envenomation should be recognized.

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