

A Case Study of Lionfish Sting-Induced Paralysis

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Abstract. The lionfish, *Pterois volitans* (Linnaeus, 1758), a venomous scorpionfish, has gained popularity among aquarium owners and has recently become established along the Southeast US, Caribbean, and Gulf of Mexico. The primary clinical effect due to envenomation is local pain, with systemic symptoms being a rare finding. Herein is reported a rare envenomation case of a 24 year old male who presented to the Emergency Department two hours following a lionfish sting to his right hand. Within three hours of envenomation he developed paralysis of all extremities. Additional symptoms included hypertension, tachycardia, and numbness of both hands. Respiratory function and range of motion of his head and neck remained intact. Hot water immersion of the affected extremity was initiated and continued throughout most of his Intensive Care Unit stay. By eight hours post-envenomation resolution of all paralysis occurred. Lionfish envenomations are typically a pain control issue and usually respond well to hot water immersion. In vitro, lionfish venom has been demonstrated to increase intracellular calcium with resulting sustained muscle contraction. Additionally, muscle fibrillation has been shown to be induced by the release of acetylcholine followed by acetylcholine depletion and loss of muscle responsiveness. These effects may explain the observed neuromuscular weakness in this patient. Lionfish envenomation has the potential to cause profound neuromuscular weakness. Treatment with hot water immersion, wound care, and good supportive measures are generally all that is required to ensure a favorable outcome. Divers and fishers should exercise caution when handling or interacting with lionfish given the potential for systematic effects from envenomation.

Key Words: lionfish, envenomation, *Pterois volitans*, paralysis, Scorpaenidae.

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Introduction

The venomous lionfish, *Pterois volitans* (Linnaeus, 1758) and *P. miles* (Bennett, 1828), native to the Indo-Pacific oceans, has gained popularity with exotic home aquaria owners (Arbuatti & Lucidi 2010). A member of the family Scorpaenidae, lionfish recently invaded the Southeast U.S. coast, the Caribbean, and is presently invading the Gulf of Mexico (Schofield 2010). Distinctive in appearance, the lionfish has 18 venomous spines located along the pelvic, dorsal, and anal fins. The primary complaint of the envenomated patient is pain at the site of the sting; occasionally radiation of pain up the affected extremity will occur. Systemic effects due to lionfish envenomation are rarely reported and are directly related to the amount of venom injected. Reported symptoms include headache, abdominal pain, hypotension, seizures, and syncope (Vetrano *et al* 2002). We report a case of lionfish envenomation leading to paralysis of all extremities.

Case Report

A healthy 24 year old male presented to the Emergency Department following envenomation by a lionfish in an aquarium. The sting was to the middle digit of his right hand and occurred approximately two hours prior to arrival. Initial findings included hypertension (154/97 mm Hg), tachycardia (109 beats per minute),

average temperature of 36.8° C, tachypnea (respiratory rate 20 per minute), and numbness of both hands. The rest of his initial neurologic examination was normal.

Within an hour of presentation, the patient lost movement in both upper and lower extremities and became diaphoretic. His ability to swallow secretions and converse with health care providers was not affected. Hot water immersion of the right extremity was initiated prior to arrival and continued throughout the majority of his hospital stay. Paralysis to all extremities continued following admission to the Intensive Care Unit (ICU).

Throughout his ICU admission, he continued to show no signs of dysphagia and retained good range of motion of the head and neck. All lab work was unremarkable. By eight hours post exposure the patient had complete resolution of all paralysis; blood pressure and heart rate returned to baseline. A urine screen was negative for any drug abuse.

Discussion

It is difficult to determine the number of lionfish exposures in the United States. Currently, the American Association of Poison Control Centers' National Poison Data System (NPDS) does not differentiate between the various types of aquatic sting exposures.



Figure 1. Lionfish (*Pterois volitans*).

A total of 1,085 fish stings were reported to the NPDS in 2007, the most recent year for which data is available (Bronstein *et al* 2008). Lionfish exposures are generally a pain control issue and are typically managed with hot water immersion (Kasdan *et al* 1987; Kizer *et al* 1985; Patel *et al* 1993).

Lionfish venom contains many heat-labile proteins; heat will denature these venom proteins, preventing them from spreading in the bloodstream and decreasing the severity of their effects. Kizer *et al* (1985) and Vetrano *et al* (2002) reported the most common symptom of a lionfish sting was pain with other symptoms included swelling, local numbness, erythema, anxiety, dizziness, nausea/vomiting, and difficulty breathing. On several occasions, lionfish stings have been attributed to deaths (Rifkin & Williamson 1996).

Some basic scientific studies have demonstrated possible mechanisms for the symptoms of a lionfish sting. Auerbach *et al* (1987) demonstrated that blister fluid from a lionfish sting contains prostaglandins and platelet aggregating factors that might contribute to the inflammatory response to the venom. The autonomic nervous system changes following envenomation were studied by Church & Hodgson (2002) and appear to be related to muscarinic and alpha receptors, mediated via nitric oxide. Further, the authors found that these effects could be diminished with antivenin for the stonefish, another member of the family Scorpaenidae, supporting the possible use of this available antivenin in serious poisonings.

In isolated muscle preparations, lionfish venom was shown to increase intracellular calcium leading to sustained muscle contraction (Church *et al* 2003). Nicotinic receptor blockade did not alter this response but calcium channel receptor antagonists potentiated the effect. These findings suggest that patients being treated with calcium channel receptor antagonists could be at higher risk for muscle contraction. They also found that stonefish antivenin neutralized the effect.

Cohen & Olek (1989) addressed the issue of neuromuscular blockade by studying an isolated preparation of frog muscle. They found that muscle fibrillation was induced by release of acetylcholine followed by depletion of this neurotransmitter and

a loss of muscle responsiveness. This may be the mechanism in human neuromuscular weakness.

Unfortunately, an electromyogram (EMG) was not performed on this patient. An EMG would have yielded valuable information which may have helped identify the cause and location of this patient's neurologic deficit. Treatment of lionfish stings is generally conservative and supportive with hot water immersion. Local digital blocks with bupivacaine help alleviate pain. The amount of venom from each spine is dependent on the depth of penetration and the amount of time the spine is left in the tissue. In the described case, our patient reportedly grabbed the lionfish, possibly placing greater pressure on the fish than typically seen with inadvertent exposures; this may have resulted in a greater than usual envenomation. There is no antivenin available; however, *in vitro* studies have shown stonefish (*Synanceia* spp.) antivenin cross reacts with components of lionfish venom (Church & Hodgson 2002; Shiomi *et al* 1989).

The use of the stonefish antivenin is not routinely recommended because of potential adverse effects and the limited toxicity of these stings. The lack of readily available antivenin is an additional problem. In patients stung by lionfish, tetanus immunization should be updated; antibiotics are not normally administered owing to the fact that the injury is from the toxin, not an infection. An additional concern is that a hypersensitivity reaction to the venom could result in future anaphylactic reactions if another sting should occur.

Conclusion

This case demonstrates the potential for a lionfish sting to produce large-scale neuromuscular weakness and paralysis. Our patient's reaction to lionfish venom apparently affected the caudal nerves first, sparing the cranial nerves much like the progression of symptoms in Guillain-Barre Syndrome and unlike the bulbar symptoms found with toxins such as botulinum. This case also displayed changes in the autonomic nervous system. As lionfish continue to invade the Southeast U.S., Caribbean, and Gulf of Mexico and gain popularity as an aquatic pet (Arbuatti & Lucidi 2010), emergency providers need to be aware that significant clinical effects from stings can be encountered. Tachycardia, hypertension, hypotension, seizures, chest pain, abdominal pain, and pain at the injection site are just some of the potential symptoms (Vetrano *et al* 2002).

Paralysis to all extremities, as reported here, is a rare but possible phenomenon. Divers should be especially cautious when interacting with lionfish, as sting-induced paralysis may be life threatening at depth. Pain management, wound care, and good supportive practices are generally all that is required in the treatment of a lionfish exposure. If paralysis is noted, monitoring of respiratory function is essential.

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