CASE REPORT

Acute Coronary Ischemia Following Centipede Envenomation: Case Report and Review of the Literature

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This is the first known case report of electrocardiographic (ECG) changes suggestive of coronary vasospasm following a centipede envenomation. A 60-year-old man presented to the emergency department (ED) 1 hour after being stung by a 12-cm centipede. He complained of right great toe pain that did not radiate to his leg. The patient had no known ischemic heart disease. He did not describe any exertional symptoms but admitted experiencing weakness. During the ED course, concurrent with obtaining peripheral intravenous access, the patient experienced diaphoresis, dizziness, hypotension, and bradycardia. His ECG showed new ST-T wave changes, which suggested an acute ischemic process. The patient's blood pressure was 89/60 mm Hg, his pulse rate was 47 beats/min, and his respiration rate was 28 breaths/min. In the following hours, ECG findings returned to baseline. His blood pressure improved gradually with fluid resuscitation after approximately 5 hours. Cardiac markers returned to normal in the 13th hour after the event, and the patient underwent exercise stress testing, which was negative. The patient was discharged with cardiology follow-up. Adult patients with centipede envenomation should be closely monitored in anticipation of possible myocardial ischemia due to vasospasm, hypotension, and myocardial toxic effects of the venom. A child receiving the same amount of venom would be potentially at greater risk.

Key words: *Scolopendra* spp, *Ototigmus* spp, centipede envenomation, electrocardiographic abnormalities, coronary vasospasm, myocardial ischemia

Introduction

We describe a patient with apparent transient myocardial ischemia associated with centipede envenomation.

Case report

A 60-year-old man presented to the emergency department (ED) 1 hour after being stung by a 12-cm centipede. He complained of right great toe pain that did not radiate to his leg. The dead arthropod was brought to the ED (Figure 1). Family history and social history were noncontributory. The patient had no known ischemic heart disease. He did not describe any exertional symptoms but admitted feeling weak. Minimal back pain triggered by movement was attributed to a musculoskeletal etiology. He was a sedentary individual and

moderately obese. The patient's medical history was significant for hyperlipidemia with a regular diet. He was on nonsteroidal anti-inflammatory drugs for rheumatoid arthritis. The patient had no known drug allergies.

On presentation, he was alert and anxious. Initial vital signs were as follows: temperature, 36.8°C; pulse, 72 beats/min; blood pressure, 150/100 mm Hg; respiratory rate, 14 breaths/min; and pulse oximetry, 96% on room air. Examination of the head and neck was normal except for uncharacteristic minimal uvular edema. No jugular venous pulsations could be seen, and no carotid bruits were heard. Cardiac examination was normal. His lungs were clear. Examination of the extremities revealed a swollen and erythematous right toe. The rest of the physical examination was within normal limits, including a nonfocal neurologic examination.

During the ED course, concurrent with obtaining peripheral intravenous access, the patient experienced diaphoresis, dizziness, hypotension, and bradycardia. His electrocardiogram (ECG) showed new ST-T-wave changes, which suggested an acute ischemic process. We

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Figure 1. The 12-cm centipede that stung the patient.

observed 1-mm inferior ST-segment elevations and 1-mm downsloping ST-segment depressions in V_1 and aVL. The T waves were inverted in V_1 and aVL, in the absence of Q waves (Figure 2). The patient was monitored immediately. His blood pressure was 89/60 mm Hg, his pulse rate was 47 beats/min, and his respiration rate was 28 breaths/min. After instituting cardiac monitoring, 300 mg of aspirin was given orally. Intravenous lines were placed, and fluid resuscitation with normal saline was begun. A chest radiograph showed no abnormalities. Intravenous heparin treatment was started pending cardiology consultation. During this time, the patient denied any chest pain.

Initial laboratory values on presentation were as follows: white blood cell count, 8.3 U/L; hemoglobin level, 13.1 g/dL; hematocrit, 38.8%; platelet count, 250 000/ μ L; Na^+ , 137 mmol/L; K^+ , 3.5 mmol/L; serum urea nitrogen, 28 mg/dL; creatinine, 1.4 mg/dL; creatinine kinase (CK), 584 U/L (reference range, 26–190); CK-MB, 9.6 IU/L (reference range, 0.0–3.9); cardiac troponin-I (cTnI), 0.5 ng/mL (reference range, 0.0–1.0); and myoglobin, 81 IU/L (reference range, 0–70). Repeat laboratory values after 12 hours were as follows: Na^+ , 137 mmol/L; K^+ , 4.2 mmol/L; serum urea nitrogen, 23 mg/dL; creatinine, 1.2 mg/dL; glucose, 89 mg/dL; CK, 515 U/L; CK-MB, 7.9 IU/L; cTnI, 0.5 ng/mL; and myoglobin, 79 IU/L. In the following hours, ECG findings returned to baseline (Figure 3). His blood pressure improved gradually with fluid resuscitation after approximately 5 hours. During this time, the patient denied any chest pain or equivalent symptoms. Cardiac Troponin-T levels were obtained on the patient in the 12th hour and were found to be normal. The patient, therefore, underwent exercise stress testing, which was negative. The patient was discharged with cardiology follow-up.

Discussion

Centipedes are elongated, multisegmented arthropods with a single pair of legs on each body segment. They are distributed widely, especially in warm, temperate, and tropic regions. Centipedes spend most of their time underground or beneath rock piles, and they usually

Figure 2. The patient's initial ECG revealed new ST-T-wave changes, which suggested an acute ischemic process.

come out at night to actively hunt their prey. The most dangerous species belong to the genus *Scolopendra*, with the largest members (*Scolopendra gigantea*) reaching lengths of 30 cm. Most species are smaller and relatively innocuous.¹ The venom apparatus consists of modified legs on either side of the body just behind the head. A venom duct carries venom to the venom claw, which is used to envenom prey via a subterminal pore on the outer curvature of the claw.²

These arthropods are fast-moving and frightening in appearance to some, and they may show aggressive behavior. However, stings usually cause no serious morbidity or mortality.³

PATHOPHYSIOLOGY

Very little scientific study of centipede venoms has been performed, but it is known that some centipede venoms are complex mixtures containing 5-hydroxytryptamine, histamine, lipids, polysaccharides, and various enzymes (eg, proteinases, esterases).⁴ Studies have demonstrated

Figure 3. In the following hours, ECG findings returned to baseline.

3 slowly moving lipoprotein bands, and the lipid components include phospholipids, cholesterol, free fatty acids, triglycerides, cholesterol esters, and squalene.⁵ A high-molecular acidic and heat-labile cardiotoxic protein (toxin S)⁴ has been isolated from the venom of *Scolopendra subspinipes*. The venom of the North American giant desert centipede *Scolopendra heros* contains a cytolysin.^{1,6}

Very little research has been performed regarding physiologic venom effects. In cats, the toxin induces a rise in blood pressure that is not influenced by α -adrenergic blockers. Muscarinic acetylcholine receptors participate in different physiologic functions (eg, reduction of heart rate, contraction of smooth muscles). An extract from the centipede *Scolopendra morsitans* has been shown to arrest the heart of the toad—an effect blocked by atropine. The mechanisms for this are unknown.⁷ Centipede venom effects on the cardiovascular system have not been studied, and the mechanism of the ECG abnormalities recorded in a few cases of arthropod stings has not been elucidated. Pulmonary edema has been observed in a few patients. Autopsy studies are very rare.⁸

In our case, we felt that the most probable diagnosis was myocardial ischemia due to vasospasm, hypotension, increased capillary permeability, inflammatory changes, and multifactorial effects of the centipede venom. It is possible that the present case could also have been related to a vagal reaction, resulting in hemodynamic compromise. This would be consistent with the absence of tachycardia in spite of hypotension.

CASES REPORTED IN THE LITERATURE

Very few data are available on the morbidity and mortality specifically attributed to centipedes. There are only 3 reported deaths due to centipedes throughout the world. Those deaths occurred in India, the Philippines, and the United States. One reported fatality occurred when a 7-year-old Filipino girl was stung on the head by a centipede of the large species *S subspinipes*, which may reach 23 cm in length.¹ A major lethal toxin (acidic protein; molecular weight, 60 000 d) derived from this species that produces vasoconstriction, increased capillary permeability, and cardiac arrest has been described.⁴ There was also a fatality in the United States from a centipede, although no details were reported.⁹

There are reports of giant desert centipede *S heros* stings causing rhabdomyolysis, resulting in acute renal failure.¹⁰ Five cases of centipede stings have been reported in Japan. The patients in these cases showed only erythema, swelling, and/or paresis around the sites.¹¹ Barnett¹² described a 6-month-old infant who ingested a centipede of the *Scutigera morpha* spp and had sys-

temic effects, which were probably due to the systemic absorption of its venom. Fortunately, the child was discharged after an uneventful course of 48 hours.

Eosinophilic cellulitis (Wells syndrome) is an uncommon condition of unknown etiology. Ilyse et al¹³ described a patient with this entity that had been triggered by a centipede sting. The presentation usually involves a vasculitis and tender cellulitis-like eruption with an infiltration of eosinophils in the dermis.

CLINICAL FEATURES

The victim typically sees the offending creature. Symptoms following envenomation may include severe pain, local tissue swelling, redness, swollen and painful lymph nodes, headache, palpitations, nausea, vomiting, and anxiety.¹ Hands and feet are the most vulnerable body parts. A local reaction, usually localized to a 10 × 10-cm area of involvement, may develop at the sting site. The wounds are typically small punctures and may exhibit small hemorrhagic vesicles.¹⁴

Mild local bleeding is common but transient.¹⁵ The site of envenomation may become ulcerated and necrotic.¹⁶ Secondary infection and local necrosis can occur.¹

The treatment of centipede stings is symptomatic and includes ice or cold compress application, simple wound care, control of pain with analgesics, and relief of pruritis with antihistamines. An infiltration of the stung area with lidocaine or another anesthetic agent promptly relieves the pain.¹⁷ Tetanus prophylaxis should be provided as indicated. In rare cases of anaphylaxis or severe toxicity, more aggressive medical treatment is appropriate. Following initial care, the wound should be examined for signs of secondary infection or necrosis.¹⁸

Conclusion

We report what is, to our knowledge, the first case of apparent cardiac ischemia following a centipede sting. This complication should be considered when treating such patients, and appropriate diagnostic studies should be obtained. A child receiving the same amount of venom might be at greater risk than an adult.

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Errata

The Collective Review by David C. Fiore, “Injuries Associated With Whitewater Rafting and Kayaking,” published in Volume 14, Issue 4, pages 255–260, contained several errors.

In Table 2 (Fatality rates), page 256, the fatality rate for Kayaking should have read: 8.7–29. The fatality rate for Driving should have read: 1.5

Therefore, the 2 sentences on the top of page 259 were incorrect and should have read as follows:

For comparison, however, in 2001, the highway fatality rate in the United States was 1.52 per 100 million miles traveled.²⁹ If we very conservatively estimate that the average distance traveled to kayak or raft is 50 miles (100 miles round-trip), that would mean the average whitewater boater has a fatality risk of dying on the highway of 1.5 per million trips, essentially within an order of magnitude of the chance of dying on the river.