

Acute ST-segment elevation myocardial infarction from a centipede bite

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ABSTRACT

Acute myocardial infarction (AMI) following a centipede bite has been very rarely reported. Here, we describe a 22 year-old man who had ST-segment elevation AMI after a centipede bite. He presented with typical chest pain, electro and echocardiographic abnormalities, and elevated cardiac enzymes with normal coronary angiography. The probable mechanisms were described. Practitioners treating centipede bites shall not consider it lightly, as centipede envenomation may produce a variety of systemic and local manifestations in susceptible individuals.

Key words: Envenomation, mechanism of action, toxins, vagal activity

INTRODUCTION

Centipedes (Class *Chilopoda*, Phylum *Arthropoda*) are slim, multisegmented arthropods with a single pair of legs per segment and one pair of antennae.^[1] The first pair of legs has been modified into two sharp stinging structures connected to muscular venom glands.^[2] They are commonly seen in warm temperate and tropical climates. They spend most of their time underground, and they are nocturnal carnivores with a wide range of prey. The species of medical importance is *Scolopendra sp*, which has been described as the longest and most the aggressive tropical centipede. Centipede venoms are complex mixtures, thus resulting in local and systemic reactions. We report one such systemic complication following centipede envenomation for its rarity, and to

create awareness among health care workers and those involved in field work.

CASE REPORT

A 22 year-old male gardener was bitten by a centipede (*Scolopendra sp*) on the radial aspect of his left middle finger while he was working. There was an immediate onset of severe localized pain and swelling. The pain engulfed the entire hand and he himself applied cold pressure on the affected parts to obtain relief. After one hour, the pain and swelling began to subside. However, with in 2 hours he began to have severe, retrosternal chest pain radiating to the left arm which was associated with sweating, nausea and vomiting. He presented to the emergency department hospital 14 hours after the bite with complaints of continued anginal chest pain. Detailed medical history did not reveal any demonstrable risk factors for coronary artery disease. He was conscious and well oriented. His blood pressure, pulse rate/minute and respiratory rate/minute were 90/60 mmHg, 74, and 22, respectively. Examination of cardiovascular, respiratory, abdominal and nervous systems was unremarkable.

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A 12-lead electrocardiogram (ECG) showed acute anterior ST-segment myocardial infarction [Figure 1]. The chest pain was not responsive to sublingual nitrates; however, subsided after intravenous morphine. Intravenous nitroglycerin, subcutaneous enoxaparin, oral clopidogrel and aspirin were given. Echocardiography showed a hypokinetic anterior wall, with a left ventricular ejection fraction of 35%. Initial laboratory tests revealed normal hemogram, electrolytes and coagulation profile. Total cholesterol(130-200 mg/dl), triglyceride(50-160 mg/dl), Low density lipoproteins (LDL)-cholesterol (less than 130 mg/dl) and High density lipoproteins (HDL)-cholesterol(35-65 mg/dl) were 117, 93, 61, and 38 mg/dl respectively. Creatinine kinase (25-192 U/L) was 831 U/L with a positive MB-fraction. Troponin I level (0-0.4 ng/mL) was markedly raised (13.2 µg/l). Emergency coronary angiography showed entirely normal coronary arteries. Three days after admission, cardiac markers were within normal limits with the resolution of electrocardiographic abnormalities; and, resolution of the anterior wall motion abnormality on echocardiography.

DISCUSSION

Centipede bites have been reported to cause constitutional and systemic symptoms^[3] including severe pain, local pruritus, headache, nausea, vomiting, anxiety, palpitations, local tissue swelling, erythema, necrosis, lymph node swelling, ischemia and rhabdomyolysis. Centipede venoms are complex mixtures containing 5-hydroxytryptamine, histamine, lipids, polysaccharides and various enzymes like proteinases, esterases etc.^[4] It is thought that the centipede venom is a lipid-toxin complex, similar to that of scorpion venom, which facilitates local cellular penetration and absorption. Very little research has been performed

regarding physiological effects of the venom. Gomes *et al*^[5] used animal models to demonstrate significant cardiovascular effects of *Scolopendra* venom, mediated by histamine and a cardiodepressant factor designated as Toxin-S, which is a high-molecular acidic and heat-labile cardiotoxic protein. Most authorities recognize the cardiodepressant toxin-S to be the lethal factor (predatory) and the histamine/serotonin components to be pain mediators (defensive).^[6] They also reported the presence of a smooth muscle contractile agent, which was recently confirmed to have muscarinic activity. Cardiac arrest in the toad after injecting the extract from the centipede *Scolopendra morsitans* was demonstrated by Mohamed *et al*,^[7] and the effect was blocked by giving atropine. The underlying mechanisms for this remain unknown.

In the English medical literature, only two cases of acute coronary syndrome after centipede envenomation have been reported.^[8,9] Even though the exact pathophysiology in both cases is unidentified, the most probable mechanisms for the present case could be acute release of inflammatory mediators inducing increased capillary permeability, inflammatory changes, hypotension and coronary artery spasm, which might have led to acute myocardial infarction with raised cardiac biomarkers. Absence of tachycardia despite hypotension, as observed in the present case, may be related to increased vagal response to centipede venom which was not documented previously in the literature.

CONCLUSIONS

This report highlights that the treating doctor shall be aware of possible serious cardiovascular complications while treating patients with a centipede (*Scolopendra sp*) bite.

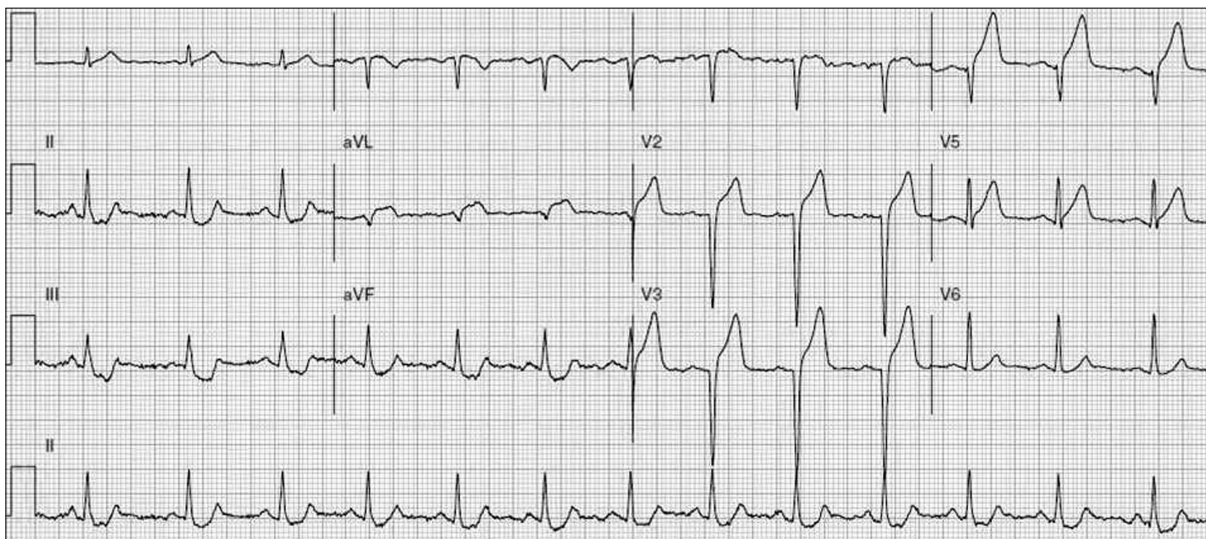


Figure 1: 12-lead electrocardiography demonstrating acute anterior ST-segment elevation myocardial infarction in the patient of the case report

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