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CASE REPORT

ST Segment Elevation Myocardial Infarction Following a *Crotalus horridus* Envenomation

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Cardiac ischemia or myocardial infarction after pit viper envenomation is rare. Few case reports have been published, none describing cases reported after crotaline snake envenomation in the United States. We report a case of ST-segment elevation myocardial infarction (STEMI) occurring in a 73-year-old man after an envenomation by a juvenile canebrake rattlesnake (*Crotalus horridus*). The man was bitten on the left index finger and subsequently developed localized edema followed by hypotension, chest pain, and altered mental status. His initial electrocardiogram revealed ST-segment elevation in the inferior and lateral leads. His hospital course included emergent left heart catheterization with thrombectomy and cardiac stent placement. This case captures the unique medical situation involving the approach to treatment and management of a patient with a severe crotaline envenomation complicated by a STEMI.

Keywords: canebrake, rattlesnake

Introduction

Crotalus horridus, commonly known as the timber rattlesnake, is regionally distributed in the northeastern, southeastern, and eastern portions of the central United States (Figure 1). Use of the common name, canebrake, prevails and refers to the southeastern timber rattlesnake.

Around 5000 venomous snakebites are reported to poison centers in the United States each year, most of which are caused by pit vipers.[1] Envenomation syndromes from timber rattlesnake bites can include local tissue effects, hematologic toxicity, and other systemic effects. Crotalidae polyvalent immune Fab antivenom therapy is available for envenomation syndromes, particularly those that entail more severe symptoms, such as progressive edema, pain, and ecchymosis from the bite site; coagulopathy, thrombocytopenia, hypofibrinogenemia, hypotension, mental status changes, and other systemic symptoms. Less commonly seen are neurotoxic effects, such as weakness, parasthesias, and altered mental status, which should also be considered in an envenomation.[2] Venom analyses have found regional populations of canebrake rattlesnakes may contain differing combinations of hemotoxic and neurotoxic factors in their venom.[3]

It is known that non-American pit viper envenomation syndromes do include myocardial ischemia, infarction, and even cardiac arrest.[4–21] In these rare cases of ST elevation myocardial infarction (STEMI), only 2 cases have been documented in the literature as undergoing percutaneous coronary intervention.[4,19] This case will outline the course and management of a North American pit viper envenomation complicated by a STEMI.

Case

A 73-year-old man was bitten by a juvenile canebrake rattlesnake on the left index finger while attempting to handle the snake at a local hunting club. The snake was captured and placed in a half-liter (20-oz) plastic bottle for identification. It had an estimated total length of 25–30 cm, but other features of the snake were not recorded. The patient rapidly developed edema, pain,

Poster presentation (Swartzentruber GS, Cibik LA, Richardson WH, Lloyd VJ. ST elevation myocardial infarction presenting as complication of *Crotalus horridus* envenomation) at the ACMT Annual Scientific Meeting, March 30, 2014, Phoenix, AZ.

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Figure 1. *Crotalis horridus* type A canebrake rattlesnake. Photograph courtesy of Scott Pfaff, Curator of Herpetology, Riverbanks Zoo and Garden, Columbia SC.

and some ecchymosis in the digit and hand. The 911 system was used, and the hunters transported the patient by private vehicle to a local landmark to meet medics. Upon arrival at the emergency department, the patient was found to be hypotensive, lethargic, and diaphoretic and reported chest pain. Intravenous fluid resuscitation and oxygen therapy was initiated during transport.

Initial vital signs in the emergency department were temperature of 36.3°C, pulse of 89 beats·min^{-1}, respiratory rate of 28 breaths·min^{-1}, blood pressure 96/72 mm Hg, and 100% oxygen saturation on non-rebreather mask. Physical examination revealed an elderly white male in moderate to severe distress. He was diaphoretic and pale with rapid shallow respirations. There was edema of the left hand with a single puncture mark on the dorsal surface of the middle phalanx second digit. Edema with some erythema was noted to include the thumb, second, and third digits and to extend proximally into the mid-metacarpal level. He was oriented to name only with verbal prompting but was otherwise disoriented, lethargic, and confused. He was given a Glasgow coma scale of 12 with a single point off for all categories. These mental status changes prevented further history from being obtained. He had dilated pupils to 6 mm and was incontinent of both bladder and bowel while in the emergency department.

An electrocardiogram obtained revealed an acute inferolateral myocardial infarction. There was 2 mm of ST segment elevation in leads II, III, and aVF and V4–6 (Figure 2). Reciprocal changes were observed in leads aVL, aVR, V1, and V2. Laboratories demonstrated a platelet count of 524 k·μL^{-1} prothrombin time of 12.3 s, international normalized ratio of 0.9, partial thromboplastin time of 28.9 s, fibrinogen 712 mg·dL^{-1}, and a point of care troponin I 0.00 ng·mL^{-1}. The patient was resuscitated with another liter of 0.9% saline followed by 5000 units of intravenous heparin, rectal aspirin 300 mg, and 6 vials of Crotalidae polyvalent immune Fab antivenom. Hypotension resolved with fluid resuscitation and subsequent blood pressures ranged from 110s to 130s mm Hg systolic. Because of his continued altered mental status and evidence of acute myocardial infarction, he was taken emergently to the cardiac catheterization suite for coronary angiography.

The cardiologist started the patient on a nitroglycerin continuous infusion. The patient was intubated in the cardiac catheterization suite by a rapid sequence intubation protocol by anesthesia due to significant hypoxemia after administration of sedative medications for left heart catheterization. During the catheterization, the patient was given eptifibatide 180 microgram·kg^{-1} bolus followed by initiation of a continuous 2 microgram·kg^{-1}·min^{-1} infusion for the next 18 hours. Cardiac angiography demonstrated a 70–80% midvessel stenosis of the circumflex artery with thrombus formation. Thrombectomy followed by primary stent implantation with a 3.5 × 23 mm XIENCE drug-eluting stent was performed with no

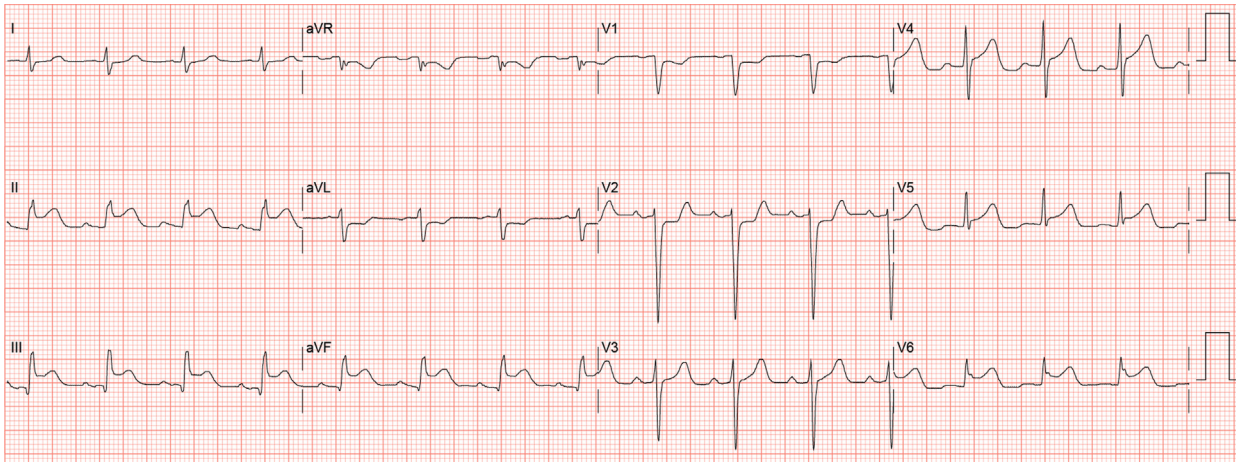

Figure 2. Initial patient electrocardiogram.