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Systemic Effects of Crotalid Envenomation Mislabeled as Anaphylaxis

To the Editor:

I read with interest the letter by Camilleri and Offerman¹ in the June 2004 issue of *Annals*. The authors describe a case of Northern Pacific rattlesnake (*Crotalus oreganus oreganus*) bite to a man's thumb. A "pressure band" was placed immediately after the bite. The patient experienced local signs and symptoms consistent with an early mild-grade envenomation, with pain and swelling limited to the distal extremity, and no signs of systemic toxicity. In the emergency department (ED), the band was removed before antivenom administration, and the patient "had rapid onset of face and neck swelling and became acutely dyspneic."

I disagree with the apparent systemic venom toxicity being labeled as anaphylaxis. True anaphylaxis is marked by signs and symptoms of massive histamine release, including itching, urticaria, flushing, bronchospasm, and hypotension. The authors do not describe these features. Anaphylaxis also requires prior antigenic sensitization.² The authors mention that the patient kept the snake as a pet, but do not comment on a history of prior envenomation or other venom exposure.

Anaphylactoid reactions to envenomation can occur but are rare. The likely cause of the patient's airway compromise was an increase in the amount of circulating venom producing systemic venom toxicity. The authors incorrectly attribute the respiratory distress to an IgE-mediated antigenic process. Increased vascular permeability with extracellular fluid leakage is a known effect of Crotalid venoms, and this likely caused the patient's angioedema.

Epinephrine, diphenhydramine, and systemic glucocorticoids were also given before antivenom. If anaphylaxis was the cause, the patient should have experienced rapid improvement.² The authors state that antivenom was started "30 minutes after intubation, once hemodynamic stability was achieved." Antivenom is the definitive treatment for severe systemic venom toxicity, and its administration should always take priority.

Precipitant systemic venom toxicity on removal of a local constrictive device has been described.³ Several Crotalid venoms, including *C. oreganus*, are known to have neurotoxic and vasoactive properties. These effects can be dramatic if

venom reaches the central circulation. It is also known that rattlesnake envenomation of the distal extremities can produce airway edema and other severe systemic venom effects.⁴

The authors correctly advise that pressure devices be maintained until the patient reaches the ED, and further suggest that antivenom be given before pressure band removal. These recommendations are found in current emergency medicine and toxicology texts.^{3,5} Additionally, when any pressure device is removed, it should be done gradually and the patient should be closely monitored. If there are any signs of systemic toxicity, the bandage should be reapplied and additional antivenom given. The authors did not mention if the band was replaced when the patient's condition worsened.

Again, I would like to stress that airway compromise is a known Crotalid venom effect and should not be confused with anaphylaxis in the absence of the other classic features of that disease process. This case does suggest that pressure devices may be beneficial in delaying systemic toxicity in Crotalid envenomations. This is a topic that we are investigating at our center, with additional information to come.

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In reply:

We appreciate the interest in our case published in the June 2004 issue of *Annals*. We have attempted to identify and respond to the issues raised by Dr. German.

Dr. German's conclusion that our reported case of acute airway edema after venom exposure was caused solely by venom effects appears to be based on a single, previously reported case by Hinze et al.¹ Actually, we believe that case bears little resemblance to ours. In that report, the envenomated patient presented with clear signs of systemic toxicity (severe hypofibrinogenemia) and had a more gradual onset of facial edema, over a 2-hour period. Our patient had no signs of systemic envenomation. Moreover, we are unable to find any previous case report describing airway edema as a direct venom effect in

the absence of other signs of systemic toxicity. In contrast, there are several reports of anaphylactic/anaphylactoid reactions to rattlesnake venom in the medical literature.²⁻⁵

Dr. German correctly notes that Crotaline venom is known to have vasoactive properties and that “these effects can be dramatic when venom reaches the systemic circulation.” It is, however, difficult to explain how our patient manifested such a serious effect as acute airway obstruction without other signs of systemic envenomation.

Dr. German asserts that, “If anaphylaxis was the cause, the patient should have experienced rapid improvement,” citing a single case report of anaphylaxis after rattlesnake envenomation.³ In that case, the patient was hypotensive at presentation and had other signs of hypersensitivity that responded quickly to administration of 2 liters of normal saline solution, epinephrine, and diphenhydramine. However, manifestations of hypersensitivity vary; the features described in that case can often be corrected quickly with standard treatment measures, whereas severe angioedema is not immediately reversible.

The statement “Anaphylactoid reactions to venom can occur but are rare” is difficult to evaluate. To our knowledge, the existing data are inadequate to establish the true incidence of anaphylactoid reactions to rattlesnake venom. Furthermore, differentiation between anaphylaxis (IgE-mediated amine release) and anaphylactoid (non-IgE mediated) in this case is impossible and clinically irrelevant.

Dr. German takes issue with the prioritization of treatment measures immediately after the onset of acute airway compromise, stating, “Antivenom is the definitive treatment for severe systemic venom toxicity, and its administration should always take priority.” The most basic tenets of emergency medicine dictate that aggressive airway intervention is the first priority for any patient manifesting acute head and neck swelling with potential airway compromise, regardless of the cause.

Airway compromise, while potentially a direct venom effect, is extremely unusual in the absence of a strike to the face or hypersensitivity reaction. The differentiation between systemic venom toxicity and hypersensitivity to venom may be difficult. We therefore recommend that envenomated patients presenting with severe, acute airway symptoms, possibly related to vasoactive amine release (regardless of IgE status), should receive airway intervention followed immediately by simultaneous treatment for envenomation and anaphylactic/anaphylactoid reaction. The inclusion of treatment measures for hypersensitivity need not delay antivenom therapy.

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IMAGES IN EMERGENCY MEDICINE

(continued from p. 94)

DIAGNOSIS:

The child reviewed here sustained an eye globe rupture from firework injury. Approximately 2,000 eye injuries occur each year in the United States from consumer firework usage, 44% of which occur in children younger than 19 years. Among the childhood cases, one third of the injuries result in permanent eye damage, and one fourth result in permanent vision loss or blindness. Ironically, the bystanders rather than the operators themselves are more frequently injured. Computed tomographic scanning is the most sensitive imaging study to detect globe rupture and to visualize the anatomy of the globe and orbit. In injuries associated with ocular hypotony and resultant scleral infolding, characteristic computed tomographic images mimic an unfolding umbrella, coined as an “umbrella sign” (Figure 2, arrow).

When globe rupture is suspected, the injured eye should be protected from any pressure with a rigid shield. Prophylactic antibiotics should also be given to prevent endophthalmitis. Ophthalmology consultation should be prompt. Surgical treatment often includes wound repair or eye globe enucleation performed on either an emergency or an elective basis within 10 to 14 days after the trauma.