

Life-Threatening Anaphylaxis Following Gila Monster Bite

*We report the case of a 40-year-old man who developed life-threatening angioedema and hypotension following the bite of a Gila monster (*Heloderma suspectum*), a venomous lizard of the southwestern United States. He recovered after treatment with IV fluids, epinephrine, antihistamines, and corticosteroids. [Piacentine J, Curry SC, Ryan PJ: Life-threatening anaphylaxis following gila monster bite. *Ann Emerg Med* August 1986;15:959-961.]*

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INTRODUCTION

The first recorded incidence of fatal anaphylaxis occurred in the year 2641 BC when King Menes died after a wasp sting.¹ Since that time, anaphylactic shock has been reported to follow exposure to a multitude of allergens. These etiologic agents range from pharmaceutical agents to human semen.²

Allergic reactions following the stings of insects are common. From 0.4% to 1% of the population may be susceptible to anaphylaxis following stings by bees, wasps, and hornets.¹ Anaphylactic shock following bites by vertebrates is rare. It has been reported following bites by rodents^{3,4} and the slow loris (*Nycticebus coucang*).⁵

We recently treated a man who suffered life-threatening anaphylaxis following a bite by a Gila monster (*Heloderma suspectum*), a venomous lizard that inhabits the southwestern United States. His clinical course, characterized by hypotension and severe angioedema of the upper airway, is reported.

CASE REPORT

A 40-year-old man with no history of asthma, atopy, or allergic reactions captured a Gila monster by placing a box on top of it. On lifting the box he sustained a bite to his left forearm. He had not been bitten by or exposed to a Gila monster previously. One-half hour later, he began to notice fullness in the throat that progressively worsened. One hour after the bite the patient presented to an emergency center complaining of shortness of breath, tightness in the throat, and drooling. He denied the ingestion of any medications or being stung by any insects. He had been drinking beer for several hours.

Initial physical examination revealed a very anxious man with marked edema of the lips and tongue who was unable to speak. The blood pressure was 100/40 mm Hg, pulse was 80, and the respiratory rate was 32. The chest and heart were normal to auscultation. No urticaria was observed. Inspection of the bite wound showed puncture wounds that penetrated skin, along with a small amount of erythema and edema.

Oxygen was administered and a 500-mL IV fluid challenge with Ringer's lactate was initiated. The patient then was transported by helicopter to St Luke's Medical Center. *En route* he received 0.3 mg subcutaneous epinephrine and a total of 100 mg diphenhydramine IV push. By the time the helicopter landed, he had received the entire 500 mL of the fluid challenge. The patient remained alert, was able to use suction to clear his own airway, and had a palpable systolic blood pressure of 86 to 90 mm Hg.

On arrival the patient was in marked respiratory distress with copious secretions that he could not control. His face was markedly edematous. Initial vital signs were as follows: blood pressure, 150/82 mm Hg; pulse, 85; and respirations, 24. His lips and tongue were swollen to three to four times



FIGURE 1. Patient on admission. Although significantly improved, angioedema of lips, tongue, and face remains.

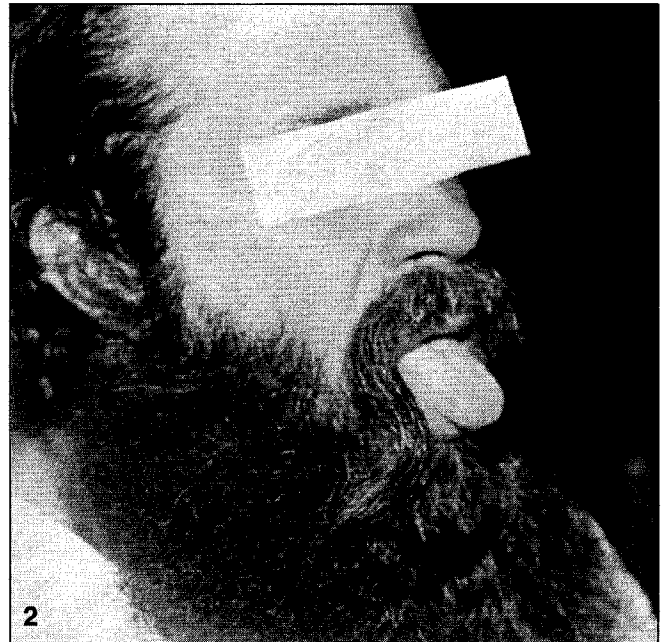


FIGURE 2. Patient just prior to discharge from hospital. Angioedema of face and upper airway has resolved.

normal size. Phonation was garbled and difficult to understand, though he was alert. The patient held his head forward in order to maintain a patent airway. His skin was diaphoretic but not cyanotic. The lungs were clear and free of any wheezing. The bite wound on his forearm consisted of small puncture wounds and a 1-cm x 1-cm area of slight erythema. Neurovascular examination of the extremity was normal. The remainder of the physical examination was unremarkable.

A decline in blood pressure from 156/82 mm Hg to 90 mm Hg systolic was treated with 0.5 mg epinephrine IV over ten minutes and with further fluid challenges so that the patient received a total of 4 L Ringer's lactate from the time of envenomation to his admission to the intensive care unit. Other treatment included 250 mg methylprednisolone IV, the inhalation of nebulized racemic epinephrine, and oxygen through nasal cannula at 4 L/minute. With this treatment his facial swelling improved somewhat and he regained control of his airway. A photograph was taken (Figure 1).

A chest radiograph and ECG were unremarkable. The patient had normal coagulation and chemistry parameters except for a serum potassium of 3.2 mEq/L. The white blood cell count was elevated at 25,000 WBC/mm³ with 71 segments and 6 bands. The hematocrit was 47.3, and the platelet count was normal at 255,000/mm³. The blood alcohol was 1.86 g/L.

The patient was admitted to the intensive care unit. To prevent further degranulation of basophils and mast cells, a low-dose epinephrine drip was started at 40 µg/hr for continual beta adrenergic stimulation and gradually was discontinued over a seven-hour period. Methylprednisolone was continued at 250 mg IV every six hours for two doses, and then 125 mg every six hours IV for two doses. Inhaled racemic epinephrine was administered one additional time, about six hours after admission.

The patient improved rapidly during the first eight hours and was transferred to a medical floor 15 hours after admission. He was observed while off of all medications, except for methylprednisolone, for an additional eight hours before discharge. Another photograph was taken just prior to discharge (Figure 2).

On telephone followup two days after discharge, the patient continued to do well and remained asymptomatic.

DISCUSSION

Anaphylactic reactions to stings by

bees, wasps, and hornets are common. Anaphylaxis following bites by vertebrates is rarely reported in the literature.

Anaphylaxis following rat bites in two laboratory workers was reported by Teasdale and Davies.³ In these workers, investigators measured specific IgE antibodies to a variety of rodent allergens including urine, dander, and serum from mice, rats, and guinea pigs. It was not noted if the workers had been bitten previously.

Wilde reported a 43-year-old man who developed hypotension, cyanosis, and tachycardia following a bite by a slow loris.⁵ The patient recovered with supportive care that included IV fluids, epinephrine, and diphenhydramine. The patient had been bitten on previous occasions. It was hypothesized that he had become sensitive to allergens in the animal's saliva.

Corkum reported a 13-year-old girl who developed moderate stridor, wheezing, and swelling of the hands and feet following gerbil bites.⁴ She also recovered after the administration of epinephrine and diphenhydramine.

The Gila monster is a venomous lizard that lives throughout southwestern Arizona and in southern Utah and Nevada. Venom is produced in a pair of venom glands in the lower jaw, and travels through ducts to the vicinity of grooved teeth. The venom is conducted by capillary action in the tooth grooves into tissue the lizard has bitten.⁶

After biting, Gila monsters may continue to hold on for as long as 15 minutes, allowing for continued flow of venom into tissue. Suggested methods to remove a Gila monster have included placing a strong stick in the back of the lizard's mouth and pushing; the application of an open flame to the underside of the animal's jaw; immersion of the extremity (and lizard) in water; and the rather unwise suggestion of grasping the lizard by the tail and jerking it from the bite.⁶

Venom from the Gila monster is complex, containing various protein components and such enzymes as serotonin, phospholipase A, protease, and hyaluronidase.⁷ Gila monster venom is not thought to affect blood coagulation.⁷

Bites by Gila monsters are rare, with most attributed to the careless handling of lizards in captivity.^{6,7} If envenomation has occurred, pain will develop within five minutes. Edema, probably due to the enzymatic activity of the venom, may appear over minutes to hours, although it usually is much less severe than the edema seen following rattlesnake bites.⁷

Occasionally, significant hypotension can occur following a bite.⁷ One of the authors has been consulted on a case in which oliguria and hypovolemia with a systolic blood pressure of 80 mm Hg developed in a 30-year-old man following a Gila monster bite to the hand. The mechanism for hypotension and shock that follows experimental envenomation of animals is not known, although Russell quotes unpublished data demonstrating a decrease in circulating blood volume after injection of venom into animals.⁷ To our knowledge, there are no documented deaths following envenomations by Gila monsters, and all victims have recovered with supportive care.

Mebs has shown that Gila monster venom released a bradykinin-like substance, and that the releasing activity of the Gila monster venom was more

potent than that of selected vipers.⁷

Our patient developed severe angioedema of the mouth, lips, and tongue following a bite by a Gila monster. The pain and mild swelling at the site of the bite indicated that he was envenomated. His hypotension may have been due to a direct effect of the venom because this has been reported previously.⁷ Although vasoactive amines might be released following an envenomation by the Gila monster, the severe angioedema seen in our patient has not been reported previously and was remote from the site of the bite. This, along with the hypotension, suggests an anaphylactic or anaphylactoid reaction from rather than a pharmacologic response to the venom or saliva of the Gila monster.

Anaphylactic reactions are characterized in part by hypotension due to a fall in plasma volume and a drop in systemic vascular resistance, urticaria, angioedema, and bronchoconstriction.¹ In treating anaphylaxis, maintaining control of the airway is of primary importance. Hypotension is treated primarily with IV fluids. Epinephrine is the drug of choice in serious anaphylactic reactions because of its ability to stimulate alpha and beta adrenergic receptors.¹ The stimulation of beta 2 receptors prevents further degranulation of basophils and mast cells, and causes bronchodilation.¹ The alpha adrenergic activity of higher epinephrine doses causes vasoconstriction and can reverse the fall in systemic vascular resistance that accompanies the severe hypotension seen in anaphylactic shock.¹ Other drugs used in the treatment of anaphylactic reactions include antihistamines, corticosteroids, theophylline, and possibly atropine. Like other patients who have suffered anaphylactic reactions following bites by vertebrates, our patient recovered using these measures.

SUMMARY

Our case suggests that there can be

allergens in the saliva or venom of various vertebrates that are capable of initiating an anaphylactic response. The allergen(s) in Gila monster saliva or venom to which our patient was sensitive is not known, especially in light of his denial of previous exposure to Gila monsters or other venomous reptiles. The treatment of anaphylactic reactions following bites by vertebrates is the same as for anaphylactic shock from other causes.

Addendum: Since the submission for publication of this article, Schmutz and Stahel⁸ have reported seven cases of anaphylactoid reactions after second, third, or fourth bites by snakes. All patients evidently had been sensitized by prior bites.

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