Bedside Management Considerations in the Treatment of Pit Viper Envenomation

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Death and disability as a result of venomous snake bite is a significant public health concern in both the United States and throughout the world. In the US, an estimated 9000 people are treated for pit viper snakebite annually,[1-3] and a death rate of 1 in 756 envenomations occurs.[4] Worldwide, an estimated 421,000 bites and 20,000 deaths occur annually from venomous snake bites, the majority of which occur in sub-Saharan Africa and Asia.[5]

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Approximately 20 types of venomous snakes are endemic to the US. All but the coral snake (family *Elapidae*) are North American pit vipers (family *Viperidae)*. The *Viperidae* subfamily *Crotalidae*, which includes the rattlesnake, copperhead, and cottonmouth (water moccasin), account for the majority of snake bites and envenomations that occur in the US.[2] Pit vipers are indigenous to every state in the US except Alaska, Maine, and Hawaii,[3] yet each snake occupies a unique geographic habitat. Copperheads and cottonmouths predominate in the southeastern US, whereas rattlesnakes have a broader geographic distribution.[4,6]

Pit Viper Characteristics

The pit viper is so named because of the heat-sensitive pit located between the eye and the nostril that senses the presence and location of warm-blooded prey or predators and guides the direction of the strike.[1] This snake has 2 large fangs, each with a hollow tip, which can inject venom locally into tissue and/or intravenously. Its distinctive triangular head (Figure 1) contains large venom glands and eyes with elliptical pupils. The rattlesnake has a rattle at the end of its tail, which provides a warning signal to a potential victim, but it does not always rattle prior to striking. A baby rattlesnake may only have a button, which is not a functional rattle.

Pathophysiology and Symptomatology of Envenomation

Snake venoms are among the most complex of all natural poisons[7] and are composed of a mixture of proteins and enzymes, including cytotoxins, neurotoxins, hemotoxins, and cardiotoxins. Venom can cause local tissue necrosis as a result of proteolysis, as well as systemic injury involving the nervous, cardiovascular, and hematopoietic systems. The quantity, lethality, and composition of venom vary with the age of the snake, the time of year, the geographic location, and the snake's diet.[1]

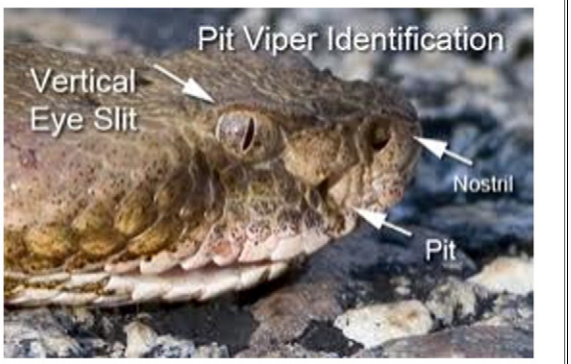

FIGURE 1
The head of a pit viper.

FIGURE 2
A finger and thumb after a snakebite. Courtesy S. Smith, MSN, RN.

NEUROTOXIC EFFECTS

Venom neurotoxins either block or excite neuromuscular junctions and can cause a variety of symptoms. Most venom neurotoxins bind to receptors, thus limiting competitive inhibition and making reversal by antivenom practically impossible. Symptoms of blocking neurotoxicity may include ptosis (drooping eyelid), drowsiness, paresthesia, respiratory distress due to flaccid paralysis of respiratory muscles (which occurs most often with Mojave rattlesnake venom), and/or an altered level of consciousness. Loss of coordination and generalized muscular weakness may occur. Mojave, timber, and Southern Pacific rattlesnake envenomations may produce a profound systemic neurotoxicity, with associated symptoms such as cranial neuropathies and flaccid paralysis.

Symptoms associated with excitation of the neuromuscular junction include muscle fasciculation or myokymia,[8] which is characterized by continuous involuntary quivering or rippling of muscles at rest as a result of spontaneous firing of groups of motor unit potentials. Fasciculations may occur in one muscle, groups of muscles, or the entire body. Antivenom may not reduce myokymia or fasciculations. Some patients who have been envenomated may describe a sensation of a metallic taste. Other systemic neurologic symptoms include dizziness, fainting, headache, and blurred vision.

VASCULAR AND HEMATOPOIETIC EFFECTS

Toxins may increase vascular permeability, resulting in extravasation of plasma and subsequent hypovolemia, and can cause effects on cardiac muscle, vascular smooth muscle, and/or other tissues.[7] Hypotension is associated with third spacing, vasodilation, nausea and vomiting, direct cardiovascular toxicity, angioedema, and neurotoxicity. Anaphylaxis may occur, and some patients may present with the signs and symptoms of severe shock. Venom procoagulant and anticoagulant enzymes exert effects on platelets, thrombinlike fibrinogenases, and prothrombin activators.[7] Anticoagulant properties may result in thrombocytopenia, hypofibrinogenemia, prolonged prothrombin time, and prolonged international normalized ratio (INR). Fibrinogen degradation and platelet aggregation and destruction may also occur.[7] Patients rarely progress to significant bleeding when treated, although cases of severe and/or fatal bleeding complications associated with hypofibrinogenemia and thrombocytopenia have been reported.[7]

LOCAL TISSUE EFFECTS

Pit viper venom causes soft tissue inflammation and necrosis (Figure 2). Symptoms include localized erythema, edema, blisters, bullae, blebs, and bleeding and/or ecchymosis[1] with associated tissue necrosis, with the potential for rhabdomyolysis and subsequent renal failure. Often the first physical sign of an envenomation is the presence of one or more fang marks and intense pain. Burning pain occurs in 90% of pit viper envenomations, although the patient may report paresthesia at the site or throughout the entire extremity. An exception is the Mojave rattlesnake bite, which causes little or no pain at the site.[1] In addition to the physical symptoms discussed earlier, patients often express a profound fear of imminent death. The signs of envenomation are summarized in Table 1.

Dry Bite

Patients who have been bitten by a snake may or may not be injected with venom. A "dry bite" is one in which fangs have