RATTLESNAKE ENVENOMATION

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Rattlesnake envenomation is a com-mon presenting complaint to southern California veterinary clinics during the summer. There are five species of crotalids, members of the pit viper fam-ily of snakes, native to this region. The most common species is Crotalus atrox, the Western Diamondback Rattlesnake, which accounts for the vast majority of local envenomation cases.

Rattlesnakes are most active during the warm months of summer and early fall (April-September) due to higher ambient temperatures and longer pho-toperiods. Rattlesnakes are most likely to be found sunning themselves early in the day, using the envi-ronment's heat to produce and store energy, and at dusk when they expend their energy stores hunting prey and mating. Adult rattlesnakes average three feet in length, are commonly found in chaparral and desert environments, and are solitary creatures. They are able to control the amount of venom inoculated into their prey and "dry" (venomless) bites are not uncommon. Juvenile rattlesnakes lack this control and are more likely to inject their full store of venom during a bite.

As a result, bites from smaller snakes are generally more dangerous than those from fully grown individuals.

Rattlesnakes hunt by stunning their prey and ingesting it whole. The composition of rattlesnake venom makes it an effective hunting tool. Vari-ous components of the venom, including proteases, thrombin-like enzymes, collagenase and phospholi-pase A, begin the process of digestion as soon as the bite is delivered. The specific make-up of a snake's venom varies by geographic location and subpopula-tion. Crotalus scutulatus, the Mojave rattlesnake, con-tains various neurotoxic compounds in its venom.

Most canine rattlesnake envenomations present after a witnessed bite or for acute onset of soft tissue swelling. The vast majority of rattlesnake bites are delivered to the patient's face or limb. Local inflam-matory responses are rapid and severe. Puncture wounds are commonly identified and develop edema, cellulitis and bruising within minutes to hours of envenomation. Tissue necrosis and sloughing may be seen, especially if the wound is allowed to prog-ress prior to the initiation of treatment. Dyspnea and upper airway obstruction may develop secondary to facial, cervical and cranial thoracic swelling, depend-ing on the location and severity of the bite.

The most common systemic effects of crotalid venom are hematologic in nature. Echinocytosis, a specific deformation of erythrocyte membranes which is easily identified on a routine blood smear, develops rapidly and can aid in confirmation of a diagnosis if the bite was not witnessed (Figure 1).

Figure 1: Blood smear showing echinocytes (spiculated red blood cells).

Coagulopathies are common and also develop rap-idly. Various components of rattlesnake venom have been shown to directly antagonize multiple coagula-tion

factors. Thrombocytopenia is also a common finding in envenomations; this results both from con-sumption of platelets in response to systemic inflam-mation and vasculitis, as well as effects of venom on megakaryocytes in the bone marrow. Many patients, especially with expedient treatment, will show evi-dence of a coagulopathy on screening blood work but will not have associated clinical signs.

Rattlesnake envenomation can cause myriad sys-temic effects and progress in a number of ways. Reported sequelae range from local inflammation to rhabdomyolysis, renal compromise, DIC, SIRS and death. Any patient presenting with a clinical suspi-cion of rattlesnake envenomation should have a com-plete physical exam performed and a minimum database collected, including blood smear and coagulation times. Systemic inflammation and distributive shock justify relatively aggressive IV fluid therapy in most envenomated patients. Blood pres-sure should be monitored, especially during the first 6-12 hours of observation. Effective analgesia should be provided promptly: standard doses of opiates such as hydromorphone are usually effective.

Antivenin therapy is the standard of care for rattle-snake envenomation in both human and veterinary medicine. Commercially available antivenins com-monly used in veterinary patients are derived from either equine or ovine serum of animals inoculated with venom of multiple species of rattlesnakes, including the Western Diamondback. Antivenin is commonly available as a dry powder which is recon-stituted and infused intravenously over a period of 1-3 hours. Specific doses and recommendations for reconstitution vary by manufacturer. Though costly, antivenin infusion is a very effective treatment, espe-cially when provided within the first 12 hours of envenomation. Type I and type IV hypersensitivity reactions have been reported with antivenin infusion and every patient should be appropriately moni-tored during treatment. Evidence of hypersensitiv-ity should be treated with antihistamines and short acting corticosteroids. Coagulation times, platelet counts, blood smears, and local signs of inflamma-tion (progression of swelling) should be reevaluated after treatment. One vial of antivenin is often suffi-cient to halt or significantly slow clinical progression, though additional vials are recommended if an ade-quate clinical response is not noted within 2-3 hours of the first infusion.

Additional supportive therapies vary significantly based on clinician preference and individual case progression. Antibiotic therapy is commonly used to prevent secondary infection of the bite wound and surrounding compromised tissue. Corticosteroids have been used for their anti-inflammatory proper-ties, however, there are no currently published stud-ies to support this practice. NSAIDs have been used, especially in cases with severe local inflammation and tissue damage; these drugs should be used judi-ciously, if at all, due to their potential adverse effects on platelet function in a patient whose coagulation may already be compromised. Serial monitoring of urine production and urinalysis is indicated if there is severe muscle damage and early treatment should be initiated if there

is any suspicion of rhabdomy-olysis or myoglobinuria. Oxygen support and airway maintenance (intubation) are sometimes required until soft tissue swelling begins to resolve.

A canine vaccine against crotalid venom is com-mercially available. The goal of vaccination is to allow the patient to form a full complement of active anti-bodies against various components of the venom, which may then be mobilized acutely in case of envenomation. As such, after the initial vaccination, a booster should be provided yearly, at the begin-ning of rattlesnake season, to maximize a positive response should envenomation occur. Vaccination is intended to minimize morbidity and does not preclude the need for immediate intervention and treatment, including treatment with antivenin. A vaccinated patient presented for rattlesnake enven-omation should be treated as aggressively as an unvaccinated patient.

There is a current lack of clini-cal studies supporting the efficacy of the canine rat-tlesnake vaccine.

Ideally, any envenomated patient should be hospi-talized and closely monitored for 24-48 hours, as it may take this long for clinical signs to fully present. Aggressive stabilization, antivenin treatment, analge-sia and close monitoring are always indicated. With prompt presentation and appropriate treatment, the prognosis for full recovery after rattlesnake enven-omation is quite good. Discharge is recommended when coagulation times, platelet counts and local wound signs appear stable, and when appropriate analgesia can be achieved with oral medications.

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