

## Spider and Scorpion Envenomation

Kate Hopper, BVSc, PhD, DACVECC

School of Veterinary Medicine, University of California, Davis, CA, USA

### Spider Envenomation

There are more than 41 000 species of spiders in the world and although almost all of them are technically venomous, only a few of them are considered medically important. The two most important clinical syndromes associated with spider bite worldwide are loxoscelism and latrodectism. There are other venomous spiders of significant medical importance but they are confined to relatively small geographical regions and will not be addressed here. Spiders of the family Theraphosidae will be briefly discussed as they can cause significant envenomation of dogs and cats.

The actual incidence of spider bites is very difficult to determine, especially in veterinary patients where it is unlikely that a spider bite can be verified. The risk of spider bites as perceived by clinicians and the lay public far exceeds the actual risk and leads to frequent misdiagnoses [1].

#### Loxoscelism

Loxoscelism describes the bite of spiders from the genus *Loxosceles*, commonly known as recluse, fiddle-back or brown spiders. There are alleged cases of necrotic arachnidism due to non-*Loxosceles* spiders, but these have not been definitively diagnosed. There are more than 100 species of *Loxosceles* spider and most are found in South America. They are endemic in the central and southern United States but are not found in the states on the east or west coasts or along the Canadian border.

The classic abnormality associated with loxoscelism is dermonecrosis. The exact pathogenesis of *Loxosceles* venom is not fully understood. Key components of the venom include phospholipases and hyaluronidase [2]. The venom can trigger an intense inflammatory

response and has direct hemolytic effects. The bite itself is not painful and human patients are often unaware that they have been bitten. This makes a definitive diagnosis challenging. It is believed that most bites by *Loxosceles* spiders cause minor erythema and edema and are self-limiting. Cutaneous loxoscelism describes the development of skin necrosis and ulceration at the bite site. Skin necrosis in human patients takes 72 hours or more to become evident. A dry necrotic eschar forms and detaches after 2–3 weeks, leaving an ulcerated lesion that can take weeks or months to heal.

Cutaneous loxoscelism is associated with non-specific systemic signs in many cases such as fever and vomiting. In contrast, systemic loxoscelism is very uncommon and is associated with intravascular hemolytic anemia developing over 7–14 days. Acute kidney injury in systemic loxoscelism is rare and has been associated with a poorer outcome in human cases [1,2].

The diagnosis is generally based on the presence of a cutaneous lesion with non-specific systemic signs in a patient with an appropriate epidemiological history. This means the patient must have been in a geographical area known to have *Loxosceles* spiders for the diagnosis to even be considered. In reality, loxoscelism is exceedingly rare. These spiders hide in dry dark places and are not aggressive; most human bites occur when the spider is trapped against the person. The likelihood of them biting animals is considered very low.

There are numerous other diseases that can cause necrotic skin lesions that may be misdiagnosed as a brown recluse spider bite. These include severe soft tissue infection (streptococcal or staphylococcal infections), pyoderma, neoplasia, toxic epidermal necrolysis, erythema multiforme, purpura fulminans, and localized vasculitis (see Chapter 137). All possible diagnoses should be investigated before considering necrotic arachnidism as a likely cause.

There are no confirmed cases of loxoscelism published in the veterinary literature. There is a case report from Brazil of a dog with necrotic skin lesions “probably” due to a *Loxosceles* spider. This lesion was treated symptomatically and the lesions took 2 months to heal [3]. There are innumerable anecdotal cases of skin lesions attributed to a spider bite in veterinary medicine; many are from geographical regions that do not have loxoscele spiders. It is likely that most of these cases are a misdiagnosis and unfortunately there is no way to determine the true incidence or nature of spider envenomation in our patients at this time. Readers are directed to Isbister and Whyte for a suggested diagnostic approach to patients with necrotic, ulcerative skin lesions [4].

Recommended first aid for a brown recluse spider bite includes elevation and immobilization of the affected limb, ice packing of the bite site, and local wound care. Supportive care is provided as indicated. Other adjunctive therapies have been suggested but none has been proven to be of benefit. Most loxoscele spider bites are self-resolving without medical intervention. Cases of severe necrosis may require surgical management but this is very uncommon and is rarely life threatening (see Chapter 166). There are conflicting reports regarding the possible benefits of glucocorticoid therapy. No detrimental effects of glucocorticoid therapy have been reported and they are commonly given to human cases of spider bite. There is no loxosceles antivenom available in the United States, although *L. lateum* antivenom is available in Mexico and South America and may be of benefit if administered within 8 hours of the bite.

The prognosis for recovery from a brown recluse spider bite is excellent; severe necrosis may lead to subsequent scarring but this is a rare occurrence [1,2].

### Lactrodectism

The widow spider group is the most medically important group of spiders in the world, causing the greatest number of human deaths. These spiders are from the genus *Lactrodectus* and are found worldwide. The venom of these spiders contains numerous toxins; the most clinically relevant toxin in mammalian envenomation is the alpha-latrotoxin. It has a unique selective effect on nerve endings, causing initial activation followed by depletion of neurotransmitters and subsequent flaccid paralysis. The female spider has fangs long enough to envenomate animals or people [2,5].

People often report little or no pain at the time of the bite but in 5–60 minutes local pain develops with increasing intensity. There may be local swelling or visible puncture marks but the major clinical sign is pain. About a third of human widow bite cases will have systemic signs, as outlined in Table 143.1. Pain is still the

dominant sign; acute abdominal pain, muscle cramps, and/or spasms are all reported. Weakness, malaise, blepharoconjunctivitis, rhinitis, and photophobia are the more common systemic signs reported in people suffering lactrodectism in North America. Infants commonly present screaming in pain and often appear to have an acute abdomen and rigidity without pyrexia. Death is rare in human medicine and usually occurs in young, old or infirm patients [2].

There is considerable species variability in susceptibility to black widow envenomation. Guinea pigs and horses are highly susceptible, cats are moderately susceptible, and dogs are relatively resistant to the effects of the venom [6,7]. The single well-described case of feline black widow spider envenomation in the literature reports acute distress with muscle stiffness and pain of the abdomen and pelvic limbs. These signs progressed to muscle weakness, flaccid paralysis, and respiratory difficulties. There was significant elevation in creatine kinase and aspartate aminotransferase. The cat was treated with a vial of antivenom and slowly improved over 5–6 days [8]. There are many anecdotal reports of black widow spider bites in dogs and cats. Most of the cases are unsubstantiated but the general clinical presentation is one of acute pain and cats tend to be more severely affected than dogs.

There is little first aid indicated for a widow spider bite; application of an ice pack may help relieve the local pain. The majority of widow spider bites in people do not require medical therapy. In more severe

**Table 143.1** Systemic signs reported in human patients with latrodectism.

Organ system	Abnormality
Cardiovascular	Bradycardia, tachycardia, arrhythmias, hypertension
Respiratory	Bronchial secretions, bronchoconstriction, pulmonary edema
Central nervous system	Psychoses, amnesia, confusion, insomnia, hallucinations, delirium
Peripheral nervous system	Pain, lacrimation, salivation, rhinitis, priapism, mydriasis, miosis
Skeletal and smooth muscle	Hypertonia, clonic contractions, fasciculations
Gastrointestinal	Nausea, vomiting, heartburn, hypersalivation, acute abdomen
Renal	Urine retention due to sphincter tone Nephritis
Hematology	Leukocytosis, neutrophilia, lymphopenia, eosinophilia, monocytosis, hemoconcentration

envenomations, supportive care should be provided, including parenteral opioid analgesia, as indicated. Antivenom therapy has been commonly recommended for patients with systemic signs, although antivenom administration is controversial as strong evidence for its efficacy is currently lacking. A randomized placebo-controlled study of widow spider envenomation in Australia found no improvement associated with antivenom therapy [5,9]. Muscle relaxant therapy with methocarbamol or diazepam has been used to treat muscle spasms.

Other therapies suggested for latrodectism include corticosteroids, magnesium, and calcium administration. Evidence for the efficacy of these therapies is scarce and they are not currently recommended in human medicine [2].

## Theraphosidae Spiders

Spiders from the family Theraphosidae are commonly known as tarantulas, bird-eating, spiders or whistling spiders. Envenomation by these spiders has minor effects in humans but can have fatal effects in animals, including cats, dogs, rats, mice, and birds. There are several cases of Theraphosidae envenomation reported in dogs in Australia. All nine cases reported died, including two dogs that were 40–50 kg in size. Many of the dogs died within 1–2 hours of being bitten [10,11]. There is limited information on the venom of Theraphosidae. As there are no reports of pets suffering tarantula bites from North or South America that the author is aware of, it is possible that members of this spider family from Australia are more venomous than other regions of the world. But given the increasing popularity of keeping Theraphosidae spiders as pets where dogs and cats may be exposed, it is important to be aware of the possible risks.

There are no specific treatment recommendations currently for Theraphosidae envenomation other than symptomatic and supportive care.

## Scorpion Envenomation

Scorpions, like spiders, are members of the class Arachnida. They have eight legs, grasping appendages at the head known as pedipalps, and a long, segmented tail. The terminal segment of the tail is a venomous stinger known as a telson. There are over 1700 species of scorpions, although only a small number of them are considered capable of causing clinically significant envenomation. Scorpions live in tropical and temperate regions of the world and pose a significant human health issue in many areas. Some of the important genera are *Centruroides*

in North America, Central America, and parts of South America, *Tityus* in South America, *Androctonus* and *Buthus* in North Africa, *Buthus* and *Leiurus* in the Middle East, and *Mesobuthus tamulus* in India. In the United States and Mexico, stings of *Centruroides sculpturatus* (bark scorpion) are potentially fatal to small children [12]. There is very little published information regarding scorpion envenomation of dogs or cats and our understanding is largely based on the human literature.

## Pathophysiology of Envenomation

Scorpion venom is a complex mixture of numerous toxic substances and there is substantial variation in composition between species. The venom can contain acetylcholinesterases, serotonin, histamine, protease inhibitors, phospholipase, hyaluronidase, and neurotoxins [7,12]. The toxins with the greatest medical importance are neurotoxins that bind to sodium, potassium, and calcium channels. Scorpion alpha-toxins bind to mammalian voltage-gated sodium channels causing membrane hyperexcitability and repetitive, uncontrolled axonal firing. The result is autonomic dysfunction and neuromuscular activity. In addition, there can be massive endogenous release of catecholamines and other vasoactive peptides, including neuropeptide Y and endothelin-1.

## Clinical Effects of Scorpion Envenomation in People

Scorpion envenomation varies in severity and clinical signs, but overall the clinical consequences are a manifestation of neurotoxic excitation syndromes leading to primarily cardiovascular and neurological abnormalities.

Pain, paresthesiae, and numbness are commonly reported in the region of the scorpion sting, soon after envenomation. Most stings only have local signs but it is possible to see systemic signs with stings of the more venomous scorpions.

Stimulation of the sympathetic nervous system can result in tachycardia, hypertension, mydriasis, restlessness, and seizures. If cholinergic stimulation is significant, it can lead to bradycardia, vasodilation, miosis, salivation, lacrimation, vomiting, and priapism. Abnormal sympathetic and/or parasympathetic tone can cause cardiac arrhythmias, including atrial tachycardia, ventricular premature contractions, ST segment changes and, less commonly, bundle branch block (see Chapter 53) [13]. Generally, the more severe envenomations are associated with predominantly sympathetic signs. Myocardial ischemia and myocarditis can occur following some species of scorpion envenomation found in Africa, the Middle East, and Asia. Neuromuscular excitation in people can be manifested as visual disturbances,

abnormal ocular movements, fasciculations, paralysis, and unco-ordinated muscular activity.

A simple grading system for evaluation of scorpion stings in human patients was recently published. A slightly abbreviated version of this grading system and general treatment recommendations is shown in Table 143.2 [13].

### Treatment of Scorpion Envenomation in People

The treatment approach to scorpion envenomation varies with the severity of the sting and a summary is provided in Table 143.2. It has been recommended that patients who present with grade 1 or 2 envenomation remain under observation for 4 hours in case of progression to a higher grade of severity. Antivenom therapy is controversial and results of studies to date have been variable. In North America, scorpion antivenom is considered beneficial but not cost-effective for routine use and it has been suggested that it should be reserved for severe envenomations [14]. In addition, the benefit of antivenom is questionable once severe systemic envenomation has been established. Benzodiazepines are

**Table 143.2** Classification and treatment of scorpion stings for human patients [13].

Clinical Grade	Clinical Effects	Potential Treatment
Grade 1	Local manifestations only	
	Pain	Analgesia
	Paresthesia	Local anesthesia
	Numbness	
Grade 2	Autonomic excitation	Antivenom
	Hypertension	Prazosin
	Agitation and anxiety	Oral benzodiazepines
Grade 3		Antivenom
	Pulmonary edema	Oxygen therapy +/- mechanical ventilation, vasodilators (e.g. prazosin)
	Hypotension & cardiogenic shock	Dobutamine, other inotropes
Grade 4	Severe neuromuscular excitation	Benzodiazepine infusion
	Multiorgan failure including coma, seizures and end organ damage due to hypotension	Antivenom
		Mechanical ventilation
		Inotropes
		Benzodiazepine infusion
		Supportive care

used in human patients to control the neuromuscular hyperactivity and opioids are commonly indicated for analgesia. Atropine administration is often avoided as, although it can alleviate many of the cholinergic signs, it may worsen the adrenergic effects. Alpha-adrenergic receptor antagonists such as prazosin and doxazosin may be of benefit to reduce some of the cardiovascular abnormalities and prevent pulmonary edema [15].

### Scorpion Envenomation in Dogs and Cats

There are numerous anecdotal reports of scorpion envenomation in dogs and some suspected cases in cats. In 2008 the Veterinary Pet Insurance company reported that scorpion stings were the fifth most common animal-induced injury to pets reported for claims.

The only published case report the author could find described scorpion envenomation (*Tityus bahiensis*) in a dog from Brazil. The presenting signs included intermittent drowsiness with bouts of vocalization and aggressiveness, tachypnea, tachycardia, and a painful region of the right thoracic limb. The animal was managed with supportive care, including a local anesthetic block around the area of the sting, and was asymptomatic by 24 hours [16].

Anecdotal reports of scorpion stings in dogs in the US are commonly described as having local signs with pain at the site of the sting but very little swelling. The *Centruroides sculpturatus* scorpion may also cause coughing. Potential reasons for scorpion envenomation to cause a cough include paresthesia of the mouth and oropharynx, as well as pulmonary edema.

Anecdotal reports of treatment of scorpion stings in dogs in the US commonly describe the use of antihistamines and glucocorticoids. There is no mention of antihistamine therapy in the treatment of human patients with scorpion stings. Given the commonly reported clinical signs of scorpion envenomation in dogs, it seems unlikely that antihistamines would be of benefit. Similarly, glucocorticoids are not used in routine therapy for scorpion envenomations in people. Studies evaluating the role of hydrocortisone in treating scorpion stings in adults and children found no benefit [17,18]. Unless there is a specific indication for their use, routine administration of glucocorticoids to treat scorpion envenomation cannot be recommended.

Analgesia is an important part of management of these cases (see Chapter 193). If severe systemic signs are present, supportive care as outlined for human patients is indicated. If severe adrenergic signs such as hypertension and tachyarrhythmias are evident, there may be a role for alpha-adrenergic receptor antagonist drugs such as prazosin (see Chapter 63). Scorpion antivenom may be beneficial in severe envenomations, but may not be

feasible in veterinary patients given the costs associated with this product.

To the author's knowledge, there are no published reports of scorpion envenomation in cats. It has been sug-

gested that cats may be more resistant to scorpion venom than dogs but there is no evidence to support this claim. It is possible that cats are more careful and agile than dogs so may not be as likely to be exposed to scorpion stings.

## References

- 1 Swanson DL, Vetter RS. Bites of brown recluse spiders and suspected necrotic arachnidism. *N Engl J Med* 2005;352(7):700–707.
- 2 Isbister GK, Fan HW. Spider biter. *Lancet* 2011;378:2039–2047.
- 3 Machado L, Antunes M, Mazini A, et al. Necrotic skin lesion in a dog attributed to *Loxosceles* (brown spider) bite: a case report. *J Venom Anim Toxins Trop Dis* 2009;15(3):572–581.
- 4 Isbister GK, Whyte IM. Suspected white-tail spider bite and necrotic ulcers. *Intern Med J* 2004;34:38–44.
- 5 Da Silva PH, da Silveira R, Appel M, et al. Brown spiders and loxoscelism. *Toxicon* 2004;44:693–709.
- 6 Brown RA. Red back spider envenomation in dogs. Control and Therapy #989. Post Grad Committee in Veterinary Science, University of Sydney, 1980.
- 7 Meier J, White J (eds). *Handbook of Clinical Toxicology of Animal Venoms and Poisons*. CRC Press, Boca Raton, 1995.
- 8 Twedt DC, Cuddon PA, Horn TW. Black widow spider envenomation in a cat. *J Vet Intern Med* 1999;13:613–616.
- 9 Isbister GK, White J. Clinical consequences of spider bites: recent advances in our understanding. *Toxicon* 2004;43:477–492.
- 10 O'Hagan BJ, Raven RJ, McCormick KM. Death of two pups from spider envenomation. *Aust Vet J* 2006;84(8):291.
- 11 Isbister GK, Page CB, Buckley NA, et al. Randomized controlled trial of intravenous antivenom versus placebo for latrodectism: the second Redback Antivenom Evaluation (RAVE-II) study. *Ann Emerg Med* 2014;64(6):620–628.
- 12 Skolnik AB, Ewald MB. Pediatric scorpion envenomation in the United States: morbidity, mortality, and therapeutic innovations. *Pediatr Emerg Care* 2013;29(1):98–103.
- 13 Isbister GK, Bawaskar HS. Scorpion envenomation. *N Engl J Med* 2014;371(5):457–463.
- 14 Armstrong EP, Bakall M, Skrepnek GH, Boyer LV. Is scorpion antivenom cost-effective as marketed in the United States? *Toxicon* 2013;76:394–398.
- 15 Bawaskar HS, Bawaskar PH. Prazosin therapy and scorpion envenomation. *J Assoc Physicians India* 2000;48(12):1175–1180.
- 16 Cardoso MJL, Sakate M, Ciampolini P, et al. Envenomation by scorpion in dog – case report. *J Venom Anim Toxins Trop Dis* 2004;10:98–105.
- 17 Bahloul M, Chaari A, Dammak H, et al. Impact of hydrocortisone hemisuccinate use on outcome of severe scorpion-envenomed adult patients. *Am J Ther* 2014;21(6):e181–188.
- 18 Bahloul M, Chaari A, Ammar R, et al. Severe scorpion envenomation among children: does hydrocortisone improve outcome? A case-control study. *Trans R Soc Trop Med Hyg* 2013;107(6):349–355.