Spider Bites

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Background: **This review provides the physician with a clinical approach to the diagnosis and management of spider bites. It examines the recent literature concerning management of bites causing dermonecrosis, secondary infection, neuromuscular damage, and allergic reactions.**

Methods: **Using the key words "spider bites," "brown recluse spider bites," "necrotic arachnidism," "black widow spider bites," "latrodectism," and "*Tegenaria agrestis* (Hobo spider)," the MEDLINE files were researched for articles pertinent to the practicing physician. Texts related to spiders and spider bites were also consulted.**

Results and Conclusions: **At least 60 species of spiders have been implicated in human bites. Most cause bites of minimal medical importance, requiring little treatment. Some (brown recluse, Hobo spider) cause severe cutaneous and systemic reactions requiring intensive medical management. The black widow bite can cause severe neurologic problems requiring the use of antivenin. Spider bites are frequently difficult to diagnose because the spider is not seen at the time of the suspected bite. Such bites should be labeled arthropod bites, vector unknown. (J Am Board Fam Pract 1995; 8:288-94.)**

Thirty thousand species of spiders identified worldwide have conquered essentially all ecological environments. Spiders, as arachnids, are closely related to scorpions, harvestmen, mites, and ticks. Approximately 60 species of spiders in North America have been implicated in human bites of medical importance. Most bites are by female spiders. Male spiders are almost always smaller and have fangs that are too short or fragile to envenomate humans. Deaths occur rarely and only with brown recluse and black widow envenomations.[1,2]

The spider's body consists of two parts. The anterior portion (cephalothorax) serves locomotion, food uptake, and nervous integration. The posterior portion (abdomen) serves digestion, circulation, respiration, excretion, reproduction, and silk production. Spiders have eight legs and six or eight eyes.[1]

Several mechanisms of injury have been described, including dermonecrosis, secondary infection, neuromuscular damage, and allergic reactions (including urticaria). Host factors also contribute to outcome. Children are more likely to have greater morbidity and mortality, hands and cutaneous areas with ample subcutaneous tissue develop more serious lesions, and individuals with underlying skin disorders develop more extensive cutaneous reactions. Treatment can also affect outcome.

The diagnosis of a spider bite is frequently very difficult to make, especially when the spider has not been seen or recovered. It is essential for medical, medicolegal, and epidemiologic reasons to be absolutely sure. The medical literature is replete with undocumented case reports and studies. In a study of 600 suspected spider bites, 80 percent were caused by other arthropods and 10 percent by other disease states (Table 1).[3]

Diagnostic requirements for spider bites include the following: (1) The basic facts about spiders place them near the bottom of biting candidates. (2) If the spider was not seen or captured close to the site of injury and at the proper time, all evidence is circumstantial. (3) Determine whether systemic arachnidism has taken place. (4) If none of the above has occurred, state "probable (or possible) arthropod envenomation, vector unknown" in the medical record.[3]

Bites of Minimal Medical Seriousness

Most spider bites involving humans cause minimal medical problems. The spider groups most commonly implicated in producing tiny cutaneous lesions include orb weavers, jumping spiders, wolf spiders, and running spiders.[1,2,4-6] Jumping spiders are the most common biter. Spiders rarely bite more than once and do not always release venom. Bites result in erythema, local edema, vesiculation, and pain. Secondary infection, ecchymosis, ulceration, and lymphadenop-

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Table 1. Different Diagnosis of Suspected Necrotic Spider Bites.

Athropods	Disease States
Kissing bugs	Erythema chronicum migrans
Imbedded tick mouth parts	Erythema multiforme
	Lyme disease
Infected flea bites	Sporotrichosis
Vesicating bettles	Stevens-Johnson syndrome
Stinging Hymenoptera	Chronic herpes simplex
Mites	Poison ivy
Bedbugs	Warfarin
Flies	Poison oak
Water bugs	Erythema nodosa
	Gonococcal arthritis dermatitis
	Diabetic ulcer
	Bedsore
	Toxic epidermal necrolysis
	Infected herpes simplex
	Periarteritis nodosa
	Pyoderma gangrenosum
	Hypersensitivity to a foreign protein

athy can occur. Forty percent of bites occur on the hands. Treatment includes cool soaks, soothing lotions, analgesics, and tetanus prophylaxis.

Loxoscelism (Brown Recluse Envenomation)

The most important necrotizing arachnid found in North America is the brown recluse. Of 13 different species of *Loxosceles* in the United States, five are associated with necrotic bites.[1] The brown recluse (*Loxosceles reclusa*) is found primarily in the south central states, with other less toxic family members scattered throughout the rest of the country. It is absent from the Pacific Northwest. The spider has a body length of 8 to 15 mm with a leg length of 18 to 30 mm. Color varies from fawn to dark brown with darker legs. There is a violin-shaped figure on the anterodorsal cephalothorax. *Loxosceles* has three pair of eyes and two segmented fangs that deliver venom (Figure 1).

The brown recluse prefers hot, dry, abandoned environments, such as wood piles, vacant buildings, rock piles, tire piles, clothes piles, and boxes. During the day it prefers a quiet place, such as a closet, beneath furniture, or in any kind of receptacle. Its natural food includes beetles, flies, moths, and other spiders, obtained mostly at night. *Loxosceles* hibernates in the fall and winter and lives 1 to 3 years. It bites defensively when trapped against the skin.[7]

An association between necrotic spider bites and the brown recluse was made in 1957. Necrotic bites were thought to be similar to those of "gangrenous spots" caused by *Loxosceles laeta* of Chile.[8] Once the relation between spider, venom, and documented bites was proved, the venom was purified. Sphingomyelinase-D was identified as the primary toxin affecting endothelial cells, red cells, and platelets. The pathological sequence involves aggregation of platelets, endothelial swelling, thrombosis, and necrosis of tissues.[9] Serum amyloid P might be required for this reaction rather than complement.[10] Severe envenomation can cause hemolysis of red cells or disseminated

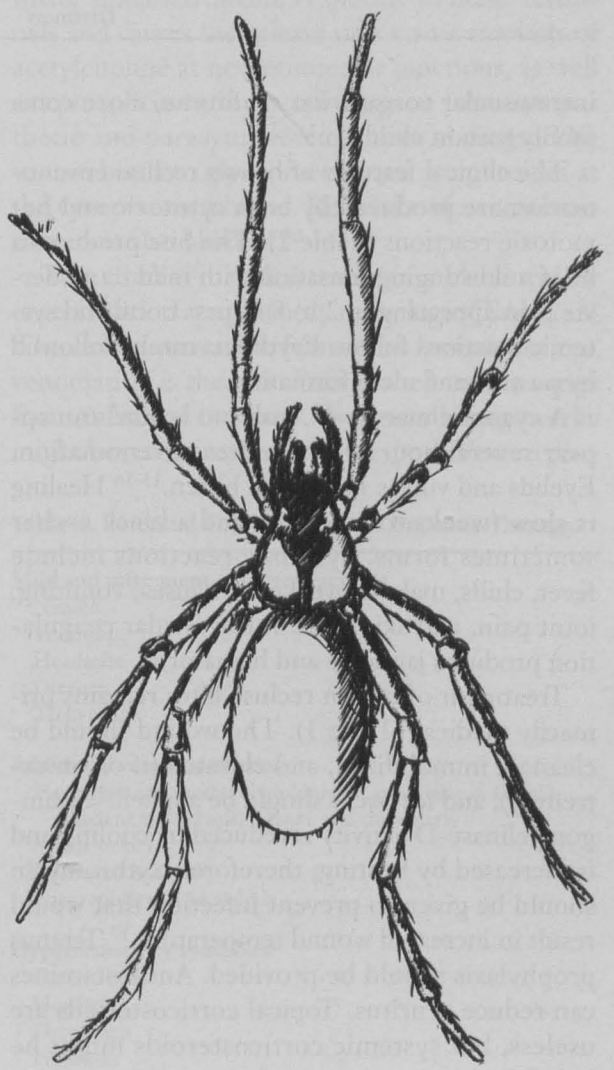

Figure 1. *Loxosceles reclusa*, the brown recluse spider — ×5.5 (5 and ½ times larger than actual size).

Table 2. Comparative Features of Brown Recluse and Black Widow Spider Envenomations.

Characteristics	Brown Recluse	Black Widow
Bite	Brief stinging sensation	Minimally painful
Pain	Moderate 2–8 hours	Intense, 15 minutes–4 hours
Tissue reaction at bite site	Necrotic ulcer Black eschar	None
Muscle reaction	None	Muscle fasciculation and spasm
Systemic reaction	Fever and chills Weakness and malaise Nausea and vomiting Joint pain Skin rash Jaundice Hematuria Disseminated intravascular coagulation	Fever and chills Sweating Salivation Urinary retention Priapism Nausea and vomiting Ptosis Headache Hypertension Dizziness

intravascular coagulation syndrome, more commonly seen in children.[11,12]

The clinical features of brown recluse envenomation are produced by both cytotoxic and hemotoxic reactions (Table 2). The bite produces a brief mild stinging sensation with mild to moderate pain appearing in 2 to 8 hours. Local and systemic reactions follow. Erythema can be followed by pustule and ulcer formation.

A cyanotic macule or "volcano lesion" can appear several hours or days after envenomation. Eyelids and vulvae have been bitten.[13-16] Healing is slow (weeks to months), and a black eschar sometimes forms. Systemic reactions include fever, chills, malaise, weakness, nausea, vomiting, joint pain, and skin rash. Intravascular coagulation produces jaundice and hematuria.

Treatment of brown recluse bites remains primarily medical (Table 3). The wound should be cleaned, immobilized, and elevated (if on an extremity), and ice packs should be applied.[7] Sphingomyelinase-D activity is reduced by cooling and is increased by heating; therefore, erythromycin should be given to prevent infection that would result in increased wound temperature.[17] Tetanus prophylaxis should be provided. Antihistamines can reduce pruritus. Topical corticosteroids are useless, but systemic corticosteroids might be helpful with severe systemic complications, including hemolysis. Immediate total wound excision should be avoided to reduce morbidity.[18,19] Delayed excision of the eschar might be necessary to allow for skin grafting. Hyperbaric oxygen therapy has been used successfully in some centers.[20,21]

Recently, treatment with dapsone has generated considerable attention.[7,19,22,23] It is a member of a sulfone group of antibiotics used to treat leprosy. Dapsone appears to act by inhibiting the inflammatory response through limiting neutrophil migration into the bite site. Dapsone is used in doses of 50–200 mg/d for 10 to 25 days. It is most effective when given early in the course of wound development. Numerous side effects have been reported (Table 4) but are infrequent to rare. The hypersensitivity syndrome is not believed to be dose related and occurs within 2 to 6 weeks following discontinuation of the drug.[24,25] Serological confirmation of brown recluse envenomation and an antivenin against sphingomyelinase-D could become available in the future.[26] Improperly treated or identified brown recluse envenomation could lead to serious long-term sequelae, such as poor wound healing, repeated failure of skin grafts, chronic pyoderma gangrenosum-like reactions, chronic pain, deep vein thrombosis, and chronic hand function impairment.[18]

Tegenaria agrestis (Common Aggressive House Spider)

A common important biter and cause of a necrotic arachnidism, particularly in the Pacific Northwest, is *Tegenaria agrestis*.[27] This common aggressive house spider, or hobo spider, is a member of the funnel-web spiders (Agelenidae). It is moderately large (10 to 15 mm in length), nondescript, brown, and has a distinct herringbone stripe on its abdomen (Figure 2). It is found in human dwellings from March through December. It nests around foundations of houses and in brick and wood piles, crawl spaces beneath mobile homes, basement corners, railroad tie piles, garages, sheds, and barns. Wandering male spiders are common biters in August and September. Prey includes

Table 3. Comparative Treatment Plans for Brown Recluse and Black Widow Envenomations.

Brown Recluse	Black Widow
Ice pack to bite site	Ice pack to bite site
Tetanus prophylaxis	Tetanus prophylaxis
Erythromycin	Calcium gluconate
Dapsone	Diazepam or meperidine
Corticosteroids for systemic complications	Antivenin
Delayed excision of eschar	
Skin grafting	

sowbugs, earwigs, silverfish, cockroaches, carpet beetles, and other spiders.

This spider was first described by Walckenaer in Europe in 1802.[28] It arrived in the Pacific Northwest in 1936, and slowly made its way across Washington state and into surrounding states. The hobo spider is the most common large spider in these areas and is the most common cause of necrotizing arachnidism. *Loxosceles* does not exist there.

The bite of *Tegenaria agrestis* is considerably more serious than that of the other small spiders. The cutaneous bite might not be felt.[29] An area of induration surrounded by erythema forms, then progresses to vesiculation, ulceration, and eschar formation. Systemic manifestations can include lethargy, headache, visual disturbances, anorexia, nausea, muscle weakness, and hallucinations. Thrombocytopenia and hemolysis can occur. About one-half of individuals envenomated develop systemic symptoms. These symptoms and findings suggest a bite similar to that of the brown recluse. It is not known whether *Tegenaria* venom contains sphingomyelinase-D. Treatment is symptomatic, and cutaneous necrosis rarely is severe enough to require skin grafting.

Latrodectism (Black Widow Spider Envenomation)

The black widow spider, *Latrodectus* species, provides an entirely different picture. The female spider, the primary envenomator, is coal black with hourglass-shaped markings of red or yellow on the ventral surface of the abdomen (Figure 3). She is subject to considerable variation in color, and several geographic species are found throughout the United States. This spider is found in every state and is more common in the south and west. She is a shy, sedentary, largely nocturnal animal that seldom leaves her silken home. The male spider is an unimportant health threat. The black widow builds an irregularly shaped mesh close to the ground with a strong-walled funnel-shaped retreat. Its common habitats include warm and dry environments both indoors and out. Webs are found under stones, logs, and debris and in corners of abandoned and infrequently used buildings. The spider hangs upside down in the web waiting for prey to become entangled. It quickly attacks, paralyzes the prey, and then sucks the juices out. If the spider is trapped against the skin or crushed, it will bite.[1]

The venom of the black widow is an oily yellow fluid containing at least 15 proteins. A neurotoxin, alpha-latrotoxin, is specific to nerve terminals and causes the release of massive amounts of acetylcholine at neuromuscular junctions, as well as epinephrine and norepinephrine from sympathetic and parasympathetic nerve endings. The venom binds to glycoproteins and gangliosides at the synaptic membrane. Reuptake of neurotransmitters is also blocked.[30]

The clinical manifestations of a black widow envenomation are primarily neurologic (Table 2).[31] The least impressive feature of *Latrodectus* envenomation is the bite. Pain at the bite site varies from minimal to sharp. Two small fang marks might be recognized as tiny red spots. Venom pro-

Table 4. Potential Complications of Dapsone Therapy.

Mild and infrequent adverse reactions
Nausea
Vomiting
Headache
Dizzines
Tachycardia
Additional rare side effects
Hemolysis and methemoglobinemia, especially in G6PD-deficient individuals, infants, and the elderly
Minor rashes
Erythema nodosum
Toxic epidermal necrolysis
Hypersensitivity syndrome
Fever
Headache
Dermatitis
Hepatitis
Hemolytic anemia
Leukopenia
Mononucleois

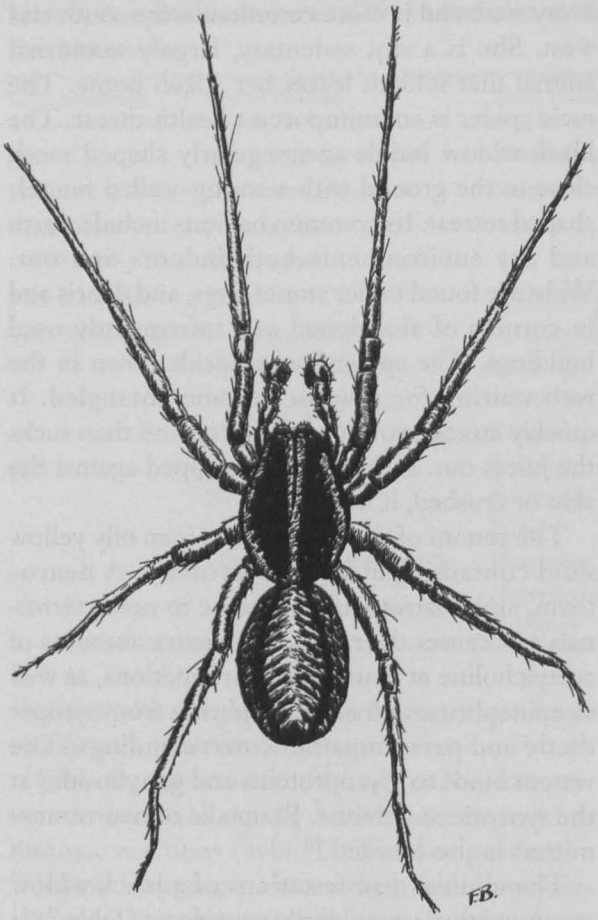

Figure 2. *Tegenaria agrestis*, the common aggressive house spider. Note the distinctive herringbone stripe on its abdomen — ×10 (10 times larger than actual size).

duces no tissue reactions. Within 15 minutes to 4 hours, muscle fasciculations and spasm begin around the bite site and then spread to regional muscles. Pain is intense and peaks in 2 to 3 hours. It can last 12 to 48 hours. Paresthesias and cutaneous hyperesthesia reinforce the diagnosis. Autonomic stimulation can produce sweating, increased salivation, fever, chills, urinary retention, priapism, nausea, vomiting, ptosis, headache, hypertension, and dizziness. Reflexes can become hyperactive. Elevated white cell count, proteinuria, and hematuria complicate the diagnosis of an acute abdomen. Acute severe hypersensitivity reactions can occur (less than 1 percent) with paralysis, hemolysis, renal failure, and coma. Delayed hypersensitivity can occur 2 to 3 days postenvenomation and cause intense pruritus with or without ecchymosis.

Treatment of black widow spider envenomation is primarily symptomatic, focusing on relieving muscle spasm (Table 3).[31] The wound should be cleaned and ice intermittently applied to relieve spasm. Tetanus prophylaxis should be given. Neurologic symptoms respond to a slow intravenous infusion of 10 percent calcium gluconate solution. A 2- to 3-mL intravenous bolus is followed by increasing doses of 2-mL increments titrated to symptoms. Cardiac monitoring is essential. The pediatric dose is 0.1 mg/kg. Doses are repeated at 2- to 4-hour intervals. Methocarbamol can be given (1 g over 5 minutes with a second gram at 100 mg/h) if calcium treatment fails. Diazepam and narcotics can also relieve symptoms but can depress respiratory drive. Dantrolene sodium, a direct-acting muscle relaxant, has been used successfully.[32] Severe hypertension is treated with standard intravenous drips of nitroprusside. Treatment with black widow anti-

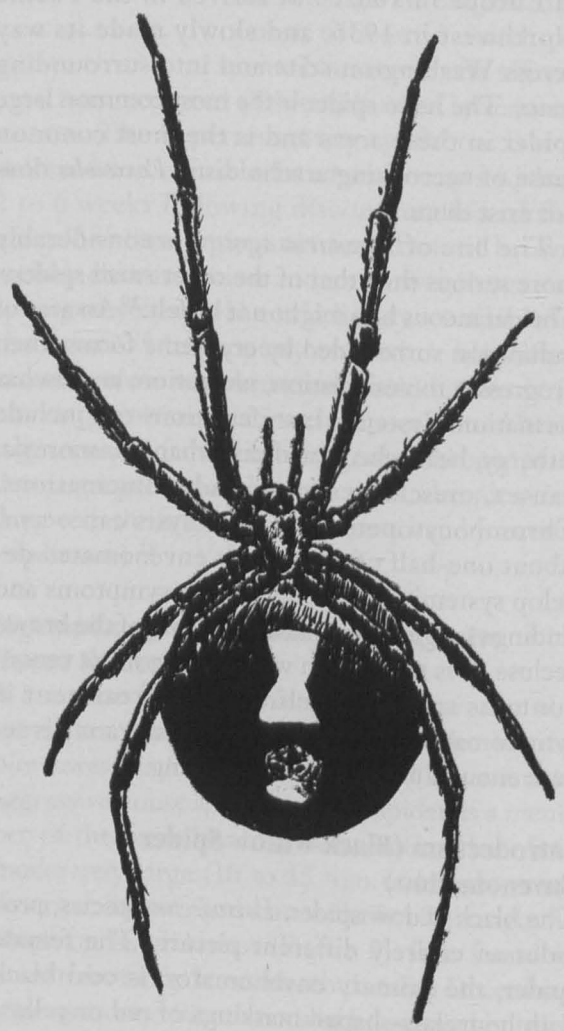

Figure 3. *Lactrodectus* species, the black widow spider. Note the hourglass-shaped marking on the ventral surface of the abdomen — ×10 (10 times larger than actual size).

venin (Lyovac-Merck, Sharpe, & Dohme) could be indicated and is quite effective. Antivenin use is generally restricted to severe poisonings and in pregnant women and children.[33,34] Obligatory skin testing is advised before a vial of antivenin (2.5 mL diluted with 10 to 50 mL of normal saline) is given. A second vial is seldom necessary. Following infusion of antivenin, relief is often dramatic and rapid (1 hour). The patient should be observed for development of acute anaphylaxis and delayed serum sickness.

Tarantulas

American tarantulas (15 to 18 cm) are not true tarantulas, but are wolf spiders. Approximately 30 to 40 species live in the United States, primarily from the Southwest United States to the Mississippi River, and then north into California, Oregon, Utah, and Southwest Idaho.[34] Tarantulas live in burrows and hunt only a few yards from their home. They attack only when vigorously provoked or roughly handled. The bite can vary from almost painless to a deep throbbing pain lasting up to 1 hour. The venom injected is primarily a hyaluronidase and a protein toxic to cockroaches and mice. Treatment includes immobilization, elevation, systemic analgesics, and tetanus prophylaxis. An unusual component of tarantula toxicity is produced from urticaria-producing hairs on the surface of the abdomen. The tarantula, when upset, will roughly scratch the lower surface of its abdomen with its legs and flick hairs into the invader's skin. The hairs cause pruritus and hives that can last several weeks. These cutaneous manifestations are particularly noted for imported color species. Treatment includes topical corticosteroids and antihistamines. If pruritus is severe, oral corticosteroids should be given for 1 to 2 weeks.

Prevention

Because spiders have little defense against insecticides, they can be reduced in numbers. Black widow spider webs can be sprayed directly. Exterminators spray or dust insecticides indoors in all large cracks and crevices, behind and under appliances, sinks, baseboards, cupboards, closets, and especially attics. Outdoors they spray under eves, around bases and window areas of houses, and in wood piles. Repeated treatment is usually necessary. It is helpful to clear away old furniture, tires, junk, newspapers, old clothes, and boxes and to plug openings and crevices into houses. Complete eradication is impossible and should not be expected.[34,35]

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