Spider Bites: Assessment and Management

by

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The members of the Committee appointed to examine the non-thesis project of JENNIFER ANNE WATSON find it satisfactory and recommend that it be accepted.

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Abstract

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In the United States, most spider bites can be attributed to the black widow
(Lactrodectus mactans), the brown recluse spider (Loxosceles reclusa), and the hobo
spider (Tegenaria agrestis). Identification of these spiders, classification of bites, biting
tendencies, and prevention of spider bites will be discussed. Differential diagnosis of
necrotic arachnidism including systemic effects and management of spider bites is included
in this paper.
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SPIDER BITES: ASSESSMENT AND MANAGEMENT

INTRODUCTION

In the United States, most spider bites can be attributed to the black widow (Lactrodectus mactans), the brown recluse spider (Loxosceles reclusa), and the hobo spider (Tegenaria agrestis). Approximately 50% of all spider bites are caused by the black widow (Hillyard, 1994). Gertsch, 1979, reports approximately 400 bites are brought to the attention of health care professionals annually in southern California. The purpose of this paper is to discuss the assessment and treatment of spider bites.

IDENTIFYING TRAITS OF THE SPIDERS

The female black widow spider is approximately 3/8” long (No-pest.com, 1999); or “smaller than a thumbnail” (Hillyard, 1994). Her body shape is globular, grape-like, and shiny jet-black with a red hourglass or anvil shape on the bottom of her abdomen (Gertsch, 1979; Hillyard, 1994; No-pest.com, 1999). A male black widow spider’s body is 1/8” long (No-pest.com, 1999), has small mouth parts, and has the reputation of never biting (Yates, 1999). Both male and female black widow spiders have much longer legs in proportion to the body (No-pest.com, 1999). The range of black widow spiders includes the northern New England states to Florida, California, Texas, Oklahoma, and Kansas (Gertsch, 1979; No-pest.com, 1999).

The brown recluse spider, also called violin spider (Gertsch, 1979) and fiddleback spider (Kelly & Schneiderman, 1995) is small to medium size and very ordinary-looking. On close inspection, one notices 3 pairs of eyes; most spiders have 4 pairs (Simplenet, 1999). Brown recluse inhabit houses (Gertsch, 1979; Hillyard, 1994) and during the night
often crawl into clothes and bedding (Hillyard, 1994). Reclusa means “seclude one” or “recluse”. As its name implies, it avoids sunlight and areas of activity; bites are rare unless the brown recluse is threatened (Clowers, 1996). Brown recluse spiders are primarily located in the southern United States (Hillyard, 1994), with the most abundant infestations in Arkansas, Missouri, Kansas, and Oklahoma (Clowers, 1996). Because brown recluse spiders can live in old boxes and furniture, they can easily be transported by humans to other areas of the country (Simplenet, 1999).

The hobo spider, also termed “aggressive house spider” (*Tegenaria agrestis*), is a venomous spider which can cause necrotic arachnidism. It is from the family Agelenidae which is indigenous to western Europe and was introduced into the northwestern United States (Port of Seattle) sometime before the 1930’s. In Europe, its primary residence is in fields, rarely entering human habitations due to the presence of major competitors; primarily the giant house spider, *T. gigantea*. In 1968, the hobo spider became established in the United States as far east as Moscow, Idaho, and as far south as Corvallis, Oregon (Vest, 1989).

In the late 1960’s and early 1970’s physicians in Idaho, Oregon, and Washington were presented with cases of probable spider bite, that closely resembled bites caused by the brown recluse spider, *Loxosceles reclusa*. Studies conducted between 1974 and 1982 suggested that another spider was probably the actual agent of these evenomations (Vest, 1998). In 1978, the only specimen of *Loxosceles reclusa* ever found in the Pacific Northwest (Prosser, WA) was in furniture that had arrived from Kansas (Akre & Catts, 1990). Finally, in 1983, field studies revealed that the hobo spider was the actual cause of
such bites in the northwest, and the spider's ability to produce necrotic lesions and systemic illness was demonstrated in the laboratory in 1986 (Vest, 1987).

Hobo spider poisoning is also termed tegenarism; the bite of a brown recluse is termed loxoscelism. Hobo spider bites are increasing in the Pacific Northwest according to the Centers for Disease Control and Prevention (MMWR, 1996). The brown recluse bite is very similar to the hobo spider bite; however, the Loxosceles species are not found in the Pacific Northwest. In Idaho, Oregon, and Washington, venomous spider bites usually are reported from areas with well-established populations of hobo spiders. T. agrestis spiders often are found in the homes of persons with these bites; recluse spiders have never been found in spider bite victims’ homes in these states (Vest, 1987).

The hobo spider is described as brown with gray “herringbone” or multiple chevron pattern markings on its abdomen and is of moderate to large size (about the size of a fifty-cent piece). Legs of the hobo spider do not have stripes, unlike the domestic and giant spiders. The sternal patterns of hobos have a light tan center with dark bands on the sides where the legs join, whereas domestic and giant spiders have small circles on the sides (Akre & Catts, 1990; Akre & Myhre, 1991; Vest, 1987; Vest 1989; Vest 1998).

The hobo spider rarely climbs vertical surfaces (unless very porous) and is uncommon above basement or ground levels (Akre & Catts, 1990; Akre & Myhre, 1991; Vest, 1998). It builds funnel-shaped webs in dark, moist areas such as under wood piles, in crawl spaces, and along foundations (Vest, 1987; Vest, 1989). Hobo spiders found in wash basins and bathtubs arrived there by falling down the slick porcelain surface; they did not come up through the drain (Vest, 1998).
**PREVENTION OF SPIDER BITES**

When working in crawl spaces, rock gardens, or similar situations, gloves and clothing that covers the skin such as long sleeves tucked into gloves, long pants tucked into boots, and coveralls or a jacket with a hood should be worn (PharmInfoNet, 1998; Simplenet, 1999; Vest, 1987; Vest, 1989). Rubber bands over pant legs and sleeves will minimize the possibility of a spider running up an arm or leg in a confined situation. Gloves or shoes should be checked before putting them on; this is a very common scenario for bites to occur.

Caution is advised when getting firewood or other items stored in potentially dangerous infested areas. Many snake and spider bites occur when the victim uses bare fingers to turn over wood or other objects that conceal the hidden agent. Hobo spiders like to rest under lawn ornaments, so firmly anchoring the base of these items into the soil, leaving no space between the soil and the object’s base is helpful. Gertsch (1979) recommends painting the underside of outdoor toilet seats with creosote, a crude oil insect repellent, to prevent the black widow spider from making the toilet seat her home. The black widow spider is easily killed with a broom or stick. Her web can easily be knocked down, and her round tan egg sacs are easily crushed with a stick or underfoot (No-pest.com, 1999).

Indoors, it is a good idea to carefully shake clothing out that has been stored or laid down in spider inhabited dwellings. Taking caution when moving boxes and objects stored in basements, cellars, or greenhouses is wise. Hobo spiders usually get onto beds by climbing bedspreads or other linen which touches or is very close to the floor. Keeping
the sides and the front of bedspreads at least eight inches above the floor and beds at least eight inches from walls should minimize the possibility of a spider climbing into the bed (Vest, 1998).

Screens on basement and ground floor windows and insulation strips under doors may decrease risk for spider infestation. Screening off dryer vents from the outside is recommended. Routine household cleaning, making certain to thoroughly vacuum in corners and closets, behind furniture, and underneath beds helps keep spider populations in your home to a minimum. Removing and securing the vacuum bag in a plastic bag for disposal will prevent spiders from crawling out of the cleaner.

Sticky “glue” traps are quite effective in areas that have many spiders making their way in (Vest, 1998). Chemical control may be necessary for large infestations underneath the house or basement. Due to the mobile nature of the hobo spider, it is virtually impossible to completely eradicate hobo spider populations. Long term results, regardless of the technique or chemical agent used rarely yield eradication. The application of pesticides outside in late June, when the spiders are not abundant on the surface will kill both recently hatched juveniles and soon to emerge adults.

The best and most effective, but often not appealing, long-term solution of hobo spider populations, is to let nature take its course. Competitive spider species, i.e., the giant spider, will keep hobo spiders out of their territory. The giant spider is relatively harmless and bites are rare. In Europe, where all three species of *tegenaria* originated, the hobo spider is not a problem (Vest, 1998).
CLASSIFICATION OF SPIDER BITES

Spider bites are placed into three separate categories: possible, probable, and proven. Possible spider bite applies to cases in which the primary provider thinks the bite could be a spider bite, but sufficient clinical or circumstantial evidence to support a firm diagnosis is lacking. A probable bite occurs when the clinical and/or circumstantial evidence supports the likelihood of a spider bite, but the biting spider was not recovered and positively identified. Proven spider bite applies to those cases in which the clinical and circumstantial evidence supports the diagnosis of spider bite, and the biting spider was captured and positively identified.

If bitten by a spider, it is very important to have collected the spider for correct identification. Smashing the spider isn’t helpful; simply trap the spider in a glass jar or container with a lid.

BITING TENDENCIES

Female widow spiders spend the majority of their time in retreat or lying perfectly still (Gertsch, 1979; Yates, 1999). Gertsch notes that the danger lies in that because these spiders live in abundance near man, they may accidentally be squeezed against the body in some way. For example, prime circumstances for the widow spider to come into contact with man include inside shoes, hidden in folds of clothing, or under objects in room corners. Female black widow spiders are timid and ordinarily make no effort to bite, even when subjected to all kinds of provocation (Gertsch, 1979; Simplenet, 1999; Yates, 1999); she will bite, however, in self-defense (Gertsch, 1979; Srv.net, 1999).
During the 1800s, the most frequent site of the black widow spider bite in male patients was the penis. A typical bite scenario occurred when a man used an outdoor privy (outhouse). The widow spider likes to nest and spin her web beneath the toilet seat. When her web is touched with an object (in this case a man's penis or scrotum), the spider senses that insect prey has landed on her web. She then rushes to the site and bites the object vigorously, much the same way she would a large insect (Gertsch, 1979).

Brown recluse spider bites occur when one is dressing or at night when sleeping (Hillyard, 1994; Simplicity, 1999). From 1896 to 1968, 126 cases of loxoscelism with 6 deaths occurred in the United States (Hillyard, 1994). Bites tend to occur March to October (Kelly & Schneiderman, 1995). Brown recluse bites tend to occur in poorly lighted environments, ie closets, outhouses, barns, attics, and cellars (Kelly & Schneiderman, 1995). The brown recluse usually bites only when threatened or inadvertently cornered (Kelly & Schneiderman, 1995).

Hillyard, 1994, reports that 20% of brown recluse bites occur on the face. However, the most most common and most severe bite site is the legs/hips, with approximately 75% of all loxosceles reclusa bites on extremities (Clowers, 1996).

Considered a fast moving spider (up to 1 m/sec), the hobo spider tends to bite if provoked or threatened (Vest, 1987; Vest, 1989; Vest, 1998). When disturbed inside a house, particularly when a light has just been turned on, hobo spiders most often remain stationary. When hobo spiders finally sense potential danger, they run, sometimes toward a person they really can’t see. The hobo spider is classified as “aggressive”, because it will move forward to attack when threatened. Although it will bite when pressed against skin
or tormented, some report it will not track people down and attack them (Vest, 1989; Vest, 1998). On the contrary, Akre & Myhre (1991) report a case in which “an aggressive house spider ran to and attacked a dog that was two feet away sniffing at the spider. It did not release its hold on the lip of the dog but had to be physically removed and destroyed.”

Mature hobo spiders are abundant mid-summer through fall when males, which are more venomous than females, are seeking females to mate. Most bites occur from August until the first “big freeze.” The severity of a bite is variable. It is believed that up to half of the bites by hobo spiders are “dry bites”, meaning that no venom was secreted (Akre & Myhre, 1991).

VENOM

Components of the hobo spider’s venom have not been identified. Researchers have, however, determined components of the brown recluse and black widow spiders’ venoms.

The black widow spider’s venom is a potent neurotoxin, estimated to be about 15 times as potent on a dry-weight basis as the prairie rattlesnake venom (Gertsch, 1979). The principal component is α-latrotoxin, a large protein with a molecular weight of about 130,000 (Hillyard, 1994). The widow’s venom acts to block transmission of nerve impulses to the muscles by overstimulating acetylcholine and noradrenaline. This causes rigidity and cramp via paralysis of sympathetic and parasympathetic nervous systems (Hillyard, 1994). The venom’s effects is sudden and causes severe stress on the human body. Its effect on other animals, however, is varied. Domestic cats are very susceptible
to widow spider venom. The dog is quite resistant; sheep and rabbits are entirely resistant. Horses and camels have been killed by black widow bites (Hillyard, 1994).

Brown recluse venom is composed of alkaline phosphatase, lipase, protease, hyaluronidase, and sphingomyelinase D (Clowers, 1996). Sphingomyelinase D triggers an enzymatic reaction which damages erythrocyte structure causing hemolysis. Hours before visible evidence of necrosis occurs, small capillaries touched by venom immediately coagulate and occlude. As coagulation begins, a large number of platelets are trapped at the site of evenomation and coagulation is worsened by mechanical damage to red cells inside the clots.

**DIFFERENTIAL DIAGNOSIS OF NECROTIC ARACHNIDISM**

Black widow spider bites are initially painful, like the prick of a needle, and two red puncture marks caused by the two fangs can usually be seen at the bite site (Simplenet, 1999; Yates, 1999). Sharp pain may set in, abate, or persist for hours at the bite site (Yates, 1999). The local dermal reaction consists of an area of erythema which disappears within several hours; no tissue necrosis occurs (Srv.net, 1999).

Brown recluse bites often do not hurt until 2-3 hours after the bite has occurred (Simplenet, 1999). When pain sets in, it is described as a mild, stinging, burning sensation (Hillyard, 1994). The most common local reaction is a mild, erythematous, painful, pruritic macule (Clowers, 1996; Kelly & Schneiderman, 1995). Most bites do not progress beyond this stage (Kelly & Schneiderman, 1995). When the bite site has a blue-gray macular halo, this indicates evolvement towards necrosis. Intense local pain over a period of 2-8 hours with bullae formation eventually turns into ischemic necrosis, leaving a
deep ulcer with a necrotic base (Molitor, 1998). Full-blown necrosis follows with formation of eschar and then sloughing and residual ulceration. Loxoscelism necrosis may heal slowly or not at all (Hillyard, 1994).

The hobo spider bite is initially painless (PharmInfoNet, 1998). Induration surrounded by expanding erythema measuring 5 to 15 cm will develop, as soon as within 30 minutes. Then, within 15 to 35 hours, a blister develops that ruptures leaving a serous exudate, encrusted, cratered wound, which often has underlying tissue necrosis that will slough. This necrotic lesion is “necrotic arachnidism”, or tissue death caused by a spider bite. Lesions usually heal within 45 days and can leave a permanent scar. If bitten in fatty tissue, healing can take up to 3 years.

Whenever possible, samples of spider bite lesion exudate should be obtained and cultured for bacterial growth. Spider bite lesions are generally sterile, and only occasionally develop secondary infection. Any “spider bite” that responds to antibiotic therapy is probably not a spider bite (Vest, 1998).

Delayed-type hypersensitivity and immediate-type hypersensitivity reactions to the bites of parasitic arthropods are the lesions most commonly mistaken for “spider bite” (Vest, 1998). Neither of these reactions, however, will produce deep, persistent dermal necrosis. Spider bites are usually single. Parasitic arthropods often bite and feed at one site, then move a centimeter or two and bite again leaving rows or patches of bites.

Vest (1998) notes that in the northwestern United States, tinea corporis and other mycoinfections are common differential diagnoses of spider bites. Bacterial infections of staphylococcal origin, particularly bacterial cellulitis and impetigo may exhibit both
erythematous swelling and/or open cutaneous ulcers which resemble stages of necrotic arachnidism. Cat scratch disease has also been implicated in an occasional “spider bite” case. Particularly in northwestern desert regions of the United States, tularemia may be confused with spider bite (Vest, 1998). Suspect tularemia when presentation includes pneumonia in conjunction with an ulcerative lesion and systemic illness.

Viral infections such as herpes zoster have been the actual causative agent in a number of suspected “spider bite” cases in the United States. Lyme disease should be considered as a possible differential diagnosis in areas where either the deer tick, *Ixodes dammini*, or the western black-footed tick, *Ixodes pacificus*, occur.

Other possible differential diagnoses of “spider bite” include erythema multiforme, erythema nodosum, scalded skin syndrome, and Stevens-Johnson syndrome. Probable necrotic spider bite cases which occur outside of the known range of the brown recluse or the hobo spider are best diagnosed as “probable necrotic arachnidism,” rather than applying the name of a suspected biter.

**SYSTEMIC EFFECTS OF SPIDER BITES**

The black widow spider’s venom is a potent neurotoxin which induces lactrodictism. This is manifested as severe muscle cramping and spasms, usually beginning in the large muscle masses of the legs or abdomen. Abdominal rigidity frequently occurs (Huston, 1993). The onset of muscle cramps usually begin within 15 minutes to 3 hours after the bite (Simplenet, 1999). The pain has been compared to that of acute appendicitis and childbirth (Srv.net, 1999). In colonial days, this was often mistaken for appendicitis and unnecessary surgical intervention was performed (Hillyard,
Similar severe muscle pain can occur in the back, thorax, groin (Huston, 1993), shoulder, and thigh (Simplenet, 1999).

Systemic effects of lactrodection include anxiety (Simplenet, 1999; Srv.net, 1999), profuse sweating, (Simplenet, 1999), piloerection, increased blood pressure (Srv.net, 1999), and shock (Yates, 1999). Neurological symptoms of lactrodection include nausea (Simplenet, 1999; Srv.net, 1999; Yates, 1999), vomiting, faintness, dizziness, tremors, loss of muscle tone, speech disturbances, convulsions (Srv.net, 1999) and general motor paralysis of various kinds (Srv.net, 1999; Yates, 1999).

It is rare for symptoms of lactrodection to persist beyond two days (Yates, 1999). Death from black widow spider bites can occur in a small child or elderly person (Srv.net, 1999) or if the person experiences an allergic reaction, hypertensive crisis, or respiratory compromise from the bite (Simplenet, 1999).

The most common systemic symptom of loxescelism, the syndrome induced by a brown spider bite, is fever (Clowers, 1996; Molitor, 1998). Other systemic symptoms include chills, malaise, nausea, (Clowers, 1996; Kelly & Schneiderman, 1995; Molitor, 1998) vomiting, myalgias (Hillyard, 1994, Kelly & Schneiderman, 1995; Molitor, 1998), and restlessness (Hillyard, 1994). The most common systemic sign of loxoscelism is adenopathy (Clowers, 1996). Complications of loxoscelism include thrombocytopenia, disseminated intravascular coagulation, (Clowers, 1996; Kelly & Schneiderman, 1995; Molitor, 1998), acute tubular necrosis (Molitor, 1998; Kelly & Schneiderman, 1995), and death (Kelly & Schneiderman, 1995).
Children and older adults are more prone to severe reaction or mortality (Clowers, 1996). Systemic reactions are noted to be proportional to local reactions, and vice versa (Clowers, 1996). Hillyard (1994), pp. 73-74, writes “a most horrifying case in 1993 was that of a housewife living near Los Angeles. The initial bite, diagnosed as that of a recluse spider, caused an extreme allergic reaction and she fell into a coma after developing toxic shock syndrome. She awoke 5 months later, by which time gangrene had affected all her extremities. She ended up having both arms and legs and the tip of her nose amputated.”

Systemic effects of tegenerism include severe and often prolonged headache (up to 4 months has been reported). The headache may start 30 minutes after the bite. Usually within ten hours after initial bite, nausea, weakness, fatigue, temporary memory loss, visual deficits, and fever can also occur (Srv.net, 1999). Rare systemic signs and symptoms which may preclude a fatal outcome include aplastic anemia, intractable vomiting, and profuse watery diarrhea. Deep vein thrombosis has been reported as a long-term complication.

[Insert Table 1 here]

**MANAGEMENT OF SPIDER BITES**

When a person presents with a suspected spider bite, the first diagnostic consideration is to rule out or confirm other possible causes. Initial diagnostic work-up includes CBC with platelets, BUN, creatinine, UA (Danis, 1997), and ESR (Molitor, 1998). Tetanus prophylaxis should be instituted (Clowers, 1996).
Initial management of a black widow spider bite, also termed "lactroectism", is centered around preventing cardio-pulmonary collapse. The bite site should be lower than the level of the heart. The bitten person should be kept warm and still, including no talking. He/she should focus on breathing easily. All clothing should be loosened and jewelry removed. Ice wrapped in cloth is placed on the bite site. Applying a tourniquet is not effective for blocking absorption of venom (Huston, 1993).

Hospitalization for black widow spider bite is recommended for all patients under age 14, over age 60, pregnant, or with hypertensive heart disease (Huston, 1993). Standard care includes establishment of intravenous access, administration of oxygen, and placement of a cardiac monitor on the patient. Gertsch (1979) recommends young children be held for observation for ten hours after the black widow spider bite has occurred.

The most common effective treatment is intravenous calcium gluconate; several courses of treatment are often required to abate muscle cramps and spasms (Srv.net, 1999). Robaxin (methocarbamol) is successful in cases not responding to calcium gluconate (Srv.net, 1999). Diazepam may ease abdominal cramping, as well (Gertsch, 1979). Antivenin (Lyovac), one ampule in 10 to 50 ml saline, is warranted in severe cases not responding to calcium gluconate or methocarbamol (Serv.net, 1999).

Similar to lactroectism, the treatment of choice for loxoscelism has yet to be established (Clowers, 1996). If bullae do not form within 6-8 hours of the bite, treatment is largely unnecessary other than to prevent secondary infection (Molitor, 1998). Ice applied to the bite site may inactivate the venom (Kelly & Schneiderman, 1995). For more
severe bites, antivenom given within the first 24 hours will decrease the lesion size and prevent necrosis; however, antivenom is not commercially available (Clowers, 1996).

Dapsone and corticosteroids have shown mixed results in animal and human studies (Clowers, 1996). Dapsone, a leukocyte inhibitor, prevents infiltration of the wound with leukocytes, thus decreasing erythema, induration, and necrosis. Severe side effects of dapsone include hepatitis, hemolytic anemia, and cholestatic jaundice (Clowers, 1996). Hyperbaric oxygen and transdermal nitroglycerin patches have shown anecdotal success (Clowers, 1996). Delayed surgical debridement of necrotic tissue at 6-8 weeks (Clowers, 1996) followed by skin grafting is sometimes required (Molitor, 1998).

Optimal treatment of tegenerism has yet to be established. Local reactions respond well to oral diphenhydramine (Benadryl) and alternating local heat and ice (Akre & Myhre, 1991). Anti-anxiety medications such as hydroxyzine (Atarax) may provide some comfort to nervous or emotionally upset victims of any necrotic spider bite. Antipyretics, e.g. acetaminophen or ibuprofen, for elevated temperature are indicated (Akre & Myhre, 1991). Antibiotics are indicated for secondary infection (Danis, 1997).

Systematically poisoned hobo spider bite victims may need hospitalization and intravenous corticosteroids for significant hematological abnormalities other than leukocytosis (Akre & Myhre, 1991; Vest, 1998). Typically, a tapered dose starting with the equivalent of 4 mg Decadron is beneficial, if administered no later than 24 hours after the bite and discontinued by the 7th day (Wand, 1972).

Management of the necrotic ulcer is not well delineated. Shaftan and Gardner (1974) recommend subcutaneous Regitine (phentolamine) at the site of the bite to reduce
necrosis and to surgically excise severe necrosis. If the victim is obese and is bitten in a highly fatty area, it is generally agreed that they are at high risk for development of deep, slow healing lesions (Akre & Myhre, 1991; Vest, 1998). Some authorities recommend that surgical excision wait until after the necrotizing process is completed (about 8 weeks), then split thickness skin grafts may be considered (Vest, 1998). Baldwin et al. (1988), however, recommend severe lesions be excised, as soon as possible.

Teaching includes advising the patient to clean the wound frequently and if the bite is on an extremity to keep it immobilized and rested. The spider’s venom is injected with a spreading agent that will travel further in thin blood, so taking aspirin is not advised. A follow-up visit should be scheduled, and the patient should watch the lesion closely for signs of darkening and signs of secondary infection. Lesions often get progressively worse before getting better, and the average healing time is 45 days (Akre & Myhre, 1991; Vest, 1998).

**IMPLICATIONS FOR PRACTICE**

Clowers (1996) suggests prospective ongoing studies of patients from entry into the health care system to wound healing would possibly correlate initial wound assessment with wound outcome. Optimal treatment modalities need to be identified, so standard treatment of necrotic arachnidism caused by *loxsceles reclusa* and *tegenaria agrestis* can be determined.

Medical references frequently cite brown recluse as the cause of necrotic arachnidism and do not mention the hobo spider. Due to the fact that brown recluse
spiders do not exist in the Pacific Northwest, health care practitioners need to recognize the hobo spider as the source of necrotic arachnidism in this area.

Further research of the hobo spider’s venom components are warranted to develop an anti-venom. Hobo spider bites are not reportable in any state; therefore, there are no reliable estimates of incidence or how often medical attention is sought. The poison control center does not have a specific designation for hobo spider evenomations. Designating a classification for hobo spider bites would provide accurate data of hobo spider bite incidence (Vest, 1998). Providers who have treated hobo spider bite cases are encouraged to publish case histories and observations on the topic.
REFERENCES


### Comparison of Black Widow, Brown Recluse, and Hobo Spider Bites

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<th>Brown Recluse</th>
<th>Hobo Spider</th>
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<tr>
<td><strong>Bite presentation</strong></td>
<td>Initially painful, pain may persist for hours</td>
<td>No pain until 2-3 hours after the bite.</td>
<td>Initially painless</td>
</tr>
<tr>
<td></td>
<td></td>
<td>Then mild, stinging</td>
<td></td>
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<td><strong>Local dermal reaction</strong></td>
<td>Erythema which disappears within several hours.</td>
<td>Mild, erythematous, painful pruritic macule to full blown necrosis</td>
<td>Induration with expanding erythema; then blisters and erupts, leaving a cratered wound with underlying tissue necrosis</td>
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<tr>
<td></td>
<td>No tissue necrosis.</td>
<td></td>
<td></td>
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<tr>
<td><strong>Systemic signs and symptoms</strong></td>
<td>Severe muscle cramps and spasms Abdominal rigidity Anxiety, diaphoresis, piloerection, increased BP, shock</td>
<td>Fever, chills, malaise, adenopathy</td>
<td>Severe, prolonged headache, weakness, fatigue, fever</td>
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<td><strong>Neurological signs and symptoms</strong></td>
<td>Nausea, vomiting, fainting, dizziness, tremors, loss of muscle tone</td>
<td>Nausea, vomiting, myalgias, restlessness</td>
<td>Nausea, temporary memory loss, visual deficits</td>
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<tr>
<td><strong>Duration of symptoms</strong></td>
<td>Less than 2 days</td>
<td>Necrosis heals slowly or not at all</td>
<td>Lesions usually heal within 45 days, up to 3 years if bitten in a fatty tissue area</td>
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<td><strong>Complications</strong></td>
<td>Allergic reaction, hypertensive crisis, respiratory compromise, death</td>
<td>Thrombocytopenia, Disseminated Intravascular Coagulation, Acute Tubular Necrosis, Death</td>
<td>Aplastic anemia, intractable vomiting, profuse watery diarrhea, deep vein thrombosis, death</td>
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<td><strong>Management</strong></td>
<td>Bite site lower than level of the heart Keep patient still Ice applied to site Hospitalize if &lt;14, &gt;60, HTN, pregnant IV CA++ Gluconate Robaxin, Diazepam Antivenin</td>
<td>Ice to site Antivenom within first 24 hours Dapsone Corticosteroids Delayed surgical debridement of necrotic tissue at 6-8 week</td>
<td>IV corticosteroids if hospitalized for systemic s/s SQ Regitine at site Delayed surgical debridement of necrotic tissue at 6-8 weeks</td>
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