Do Hobo Spider Bites Cause Dermonecrotic Injuries?

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In 1987, the hobo spider, Tegenaria agrestis, was initially implicated in necrotic lesions in the Pacific Northwest of the United States. ¹,² This European-origin species has since joined the widow and recluse spiders to become the well-known trio of medically significant spiders in North America. It is currently distributed from British Columbia to Oregon and east to Montana and Colorado, with eastward expansion still occurring.³ Yet, because of the spider’s notoriety, people throughout the United States assume that hobo spiders are locally present, and physicians have diagnosed bites in California where hobo spiders have never been documented.³

Worldwide, spiders have routinely been implicated in necrotic lesions. In some cases, however, these incriminations were based on poor clinical reporting, insufficient research, circumstantial evidence, or extrapolations from necrotic effects of venom in animal models. For example, in South America, wolf spiders were blamed for causing necrotic ulcers in the 1920s on the basis of suspected cases. This prompted studies in animals demonstrating that large quantities of venom injected intradermally caused necrosis, but necrosis was not caused when venom was injected subcutaneously or intramuscularly.⁴,⁵ Anti-venom was developed and used in Brazil, and wolf spiders in other parts of the world were blamed for necrotic ulcers by association.⁵ However, subsequent documentation of 515 verified bites manifested no necrotic ulcers.⁴ Unfortunately, wolf spiders are still blamed for necrotic ulcers in many parts of the world, including the United States.⁶ In Australia, the medical community rapidly accepted the white-tail spider (Lampona sp) as a major etiologic agent for necrotic arachnidism on the basis of reports of unsubstantiated cases and suspected bites without spider involvement over a 20-year period.⁷-¹⁴ A recent prospective study of 130 definite bites without necrosis provides significant evidence that white-tail spiders are very unlikely to cause necrotic ulcers.¹⁵ The hobo spider has been widely accepted as a dermonecrotic agent in North America, with this information becoming reiterated as uncontested fact in medical review articles and major textbooks.⁶,¹⁶-¹⁸ Suspected hobo spider bites have been proposed to be the predominant cause of necrotizing arachnidism where the spider is found.¹⁹ However, a critical examination of the original hobo spider envenomation literature begs for a more discriminating look at the veracity of hobo spider venom toxicity; the definitive evidence is sparse.

Vest¹ initially implicated the hobo spider as a dermonecrotic agent by pushing live spiders into shaved rabbit flesh, forcing envenomations. The resultant necrosis was considered sufficiently valid circumstantial evidence to elevate the hobo spider to that of medical significance. In little more than a decade, it was cited as such in general medical texts, frequent bite diagnoses were referred to the authors of a hobo spider distribution study,³ and requests to identify potential hobo spiders came from areas of North America far outside the hobo spider’s known range. Although animal bioassays are critical toxicology research tools, there are cases of differential mammalian toxicity that would lead to incorrect extrapolations in human beings (eg, Atrax spider bites are highly deleterious to primates but not other mammals, whereas Australian tarantula spider bites are fatal to dogs but cause little effect in human beings).²⁰

A study of 75 cases of suspected spider bites by Vest² initially implicated hobo spiders in human envenomation where he described 22 cases of “necrotic arachnidism” after excluding confirmed/suspected spider bites of other species, other arthropod bites, and cigarette burns. However, his case definition of “necrotic arachnidism” appears arbitrary and is based on the appearance of a skin lesion. Hobo spiders were then implicated because they were subsequently found in the homes of 16 of 22 patients. A
spider was confirmed biting the patient in only 4 cases (all reported no immediate pain) and in no case was the spider submitted for expert identification. Akre and Myhre list 34 case histories for potential Tegenaria spider bite, yet the only verified hobo spider bite involved a dog as the bite victim. Fisher et al presented one case where hobo spiders were discovered in the home of the patient with a necrotic lesion and, in a second case, someone walked into a spider web, felt no pain at the time, developed lesions on the hand and forearm, and saw a large brown spider (neither collected nor identified) in the area. Neither case is convincing for hobo spider involvement because hobo spiders (1) are commonly found in Pacific Northwest homes so their mere presence does not imply guilt and (2) they make webs under objects very close to the ground, making it feasibly improbable to walk into its web. In addition, hobo spiders are reticent to bite, retreat quickly with disturbance, and are sufficiently large that it is highly unlikely that the spider could both crawl on one’s exposed flesh and inflict multiple bites unnoticed. Sadler et al described a woman developing a nodule with pustules, inflammation, and 2 areas of ecchymosis after feeling a bite in bed (a spider, later identified as T agrestis, was found on the wall adjacent to the bed the next day). Proximity of hobo spider or other large unidentified brown spiders without verification of the act of biting is insufficient evidence for incrimination.

The only verified hobo spider bite in the medical literature resulting in a necrotic lesion involved a 42-year-old woman with history of phlebitis who found a crushed literature resulting in a necrotic lesion involved a 42-year-old woman with history of phlebitis who found a crushed spider in her pants after feeling a burning sensation. After 3 hours, a vesicle developed which eventually ruptured to form an ulcer, enlarging over 10 weeks after which she sought medical advice. She was prescribed antibiotics and eventually a deep venous thrombosis was diagnosed after a venogram. There is no information as to whether the ulcer was cultured for other infective agents. It is therefore unclear in this case whether or not the ulcer was the result of pre-existing venous disease, a common cause of ulceration. A search of MEDLINE (last checked December 18, 2003), using the search terms “Tegenaria” and “Hobo spider,” found no other cases of suspected or definite bites. Therefore, the evidence for hobo spider envenomation causing necrotic ulcers consists of one verified bite in a patient with a pre-existing medical condition known to cause ulcer disease, rabbit model bioassays, and many purported envenomations that could easily be ascribed to other etiologies.

Despite the questionable foundation of proof, the hobo spider’s potential for causing medical injury has been referenced in subsequent publications and transmuted into conventional wisdom by repeated citation, losing sight that the basis is presumptive or circumstantial. A variety of medical conditions of diverse causes manifesting in dermonecrosis have been misdiagnosed as necrotic spider bite. Without substantial validation of verified hobo spider envenomation effects, the medical community will misdiagnose presumptive hobo spider bites. This parallels a striking similarity to the brown recluse spider, which is erroneously blamed for necrotic lesions throughout North America, including states or regions where brown recluse are exceedingly rare or have never been found. Although hobo spiders may rarely cause necrotic lesions, it seems that, at the least, its role in North American dermonecrosis is still unproven and, at the most, the hobo spider might be erroneously implicated. Preliminary corroboration shows that hobo spiders are not considered medically significant in its native Europe; Binford found no differences in venom composition between European and American T agrestis spiders and suggested that the hobo spider has been wrongly accused of causing necrotic skin lesions.

The hobo spider toxicity syndrome needs more detailed examination using prospective studies of large numbers of definite bites. This methodology has been described in detail elsewhere but involves the patient seeing and collecting the spider immediately at bite inception and the spider subsequently being identified by an expert arachnologist. This will allow a description of the spectrum of hobo spider bite manifestations rather than relying on suspected bites, anecdotal evidence, and the publication of individual case histories of extreme manifestation, which because of rarity of expression or uncommon synergy with concurrent underlying maladies, will misrepresent the average reaction. This approach has been used in Australia to demonstrate that spiders rarely cause necrosis and that bites by the implicated white-tail spider were unlikely to cause necrotic ulcers. Although it is accurately posited that verified bite information is difficult to acquire because of the rarity of cases, we then pose the question that, after several decades of continual coexistence with hobo spider populations in densely populated human areas (e.g., Portland, OR; Seattle, WA; Salt Lake City, UT, and its suburbs), if the etiology is accurate, shouldn’t there be more definitive evidence of hobo spider involvement? A similar argument has demonstrated that recluse spiders are highly unlikely to cause many suspected cases of necrotic arachnidism reported from nonendemic areas.
Necrotic lesions of unknown etiology should not be immediately attributed to spider bites, and we vigorously appeal for validation of hobo spider incrimination lest erroneous information continue to proliferate that can only be detrimental to proper health care. Incorrect diagnosis of necrotic ulcers as spider bites (eg, basal cell carcinoma, anthrax, bacterial and fungal infections) delays or prevents appropriate treatment, sometimes resulting in significant morbidity. It will require cooperation of both medical and arachnological communities to ferret out this difficult-to-acquire information, because hobo spiders cannot be identified accurately by coloration; one must examine microscopic structures for accurate species identification, which requires significant arachnological skills. Until definitive substantiation is accomplished, it seems more logical to question the role of hobo spiders in necrotic lesions rather than to continue blindly, possibly making erroneous diagnoses on the basis of insufficient evidence. It may turn out that hobo spiders are medically significant, but considering the previous mistakes made with white-tail spiders and others, it would be prudent to require more proof. It would not be surprising if much of the currently accepted dogma regarding hobo spider bites eventually turns out to be wrong due to the largely presumpstive body of evidence that is the basis of the hobo spider envenomation syndrome, along with the myriad of nonspider dermonecrotic etiologies that can be mistaken for necrotic spider bite.

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