CLINICAL VIGNETTE

Latrodectism: A Personal Experience

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It was midsummer and my then fiancée and I drove to Palm Desert, CA. Temperatures were easily over 100 degrees. The purpose was to help my mother with her obligate home downsizing as my little sister had finally moved out. The plan was to have the movers take the large items and then load the 3 family SUVs that were there to help with the smaller fragile items. On moving day, the movers arrived and started to move the furniture. I was in the garage sorting through boxes of old memories, high school trophies, and certificates for events that I didn't even remember. The garage was hot as it was not air conditioned. Through layers of sweat and drinking watered-down Gatorade, I started loading the back of our SUV. Having grown up in the desert, I was aware that there were several disorganized webs around the sides of boxes that are known to be classic of the black widow spider. I was careful, I thought.

After loading the first trip, I started to notice my right anterior shoulder was starting to ache with a sharp pain in my axillae and side. I started to range it, trying to place when I had pulled it. Fast forward 30 minutes and the pain started to worsen. Thank goodness that the medicine drawer hadn't been emptied, and I popped a few Tylenol and Motrin. Yet, this was not helping, and as the pain continued to worsen, I took my shirt off to see where the pain originated. My whole anterior shoulder was erythematous; there was a clear puncture in the middle. At this point, I had a strong suspicion as to what was causing my pain and probably more than a few expletives ensued. I had dealt with a few cases in the ED with the same condition, and I was hoping that my course would be a mild one. It was not.

Within the hour, the abdominal and back pain started with nausea and vomiting. Clinically, I had an acute abdomen. I had total body dolor. At this point, I could not find a position to stand, so I tried to lay down. Unfortunately, the sofa and beds were gone, so I laid on the ground. That didn't work; I tried to sit then walk. I jumped in the shower, but the cold water hurt and a trial of the warm water was worse. As that failed, I again tried to alternate walking and laying with multiple stops to vomit. By this time, my fiancée was trying to convince me to go to the local ED. I refused using the argument that all that would be done is IV fluids, analgesics, and muscle relaxants. Unless I was unstable, there would be no use in the antivenom, plus I didn't want to pay the deductible. A little later the pain continued to worsen (I would say 10/10), and I agreed it was time to go to the ED. Unfortunately, the cars had been driven away as I had told the remainder of the family that it was nothing. My fiancée began to contact them so that someone could come. I adamantly refused an ambulance and told my fiancée if I became altered or unstable then paramedics would be okay. Finally, my family came. Pain was worse; I redefined what 10/10 was. I was now having tingling of both my upper and lower extremities, and it made walking difficult. I took the back seat to lay out. When I arrived to the ED, I must have looked how I felt as I was taken immediately to a bed. I was febrile and tachycardic. In my bed, I put on a gown and had the experience of being a patient in an ED. I was placed on a monitor and had an IV inserted, which was opened to normal saline. I told an abridged version of the events, and after a quick exam an antiemetic, analgesic and a muscle relaxant were administered intravenously. As my symptoms were severe, we spoke about the antivenom and decided that the risks were not worth the benefit. After several hours, the pain and vomiting had begun to improve. I was ready to be discharged. A small victory was that I did not require admission. When we got to my mom's new place, I sent an email to the ED group saying I would be unable to work as I was on the schedule the next day. In 7 years, I had never taken a sick day. I was off for a week. Ultimately, the body pains resolved, but the foot tingling remained. My fiancée drove us home to Los Angeles. I was still in the back seat as my feet tingled and hurt, and I needed to keep them up as I had found that this helped. I was not aware that the post black widow neuropathy lasts several weeks. It made working difficult as it hurt to walk. I found a new appreciation for neuropathic pain. After a few weeks, I returned to normal. When I tell the story, my wife still gets annoyed and calls me stubborn for not going to the ED sooner. After all that, no superpowers developed.

Discussion

Widow spiders (*Latrodectus* spp.) are found worldwide and cause latrodectism, a condition of localized, regional, or generalized pain with associated nonspecific symptoms with autonomic and neurologic disturbances.[1-5] European authors have reported widow bites for over 2,000 years.[1]

Widow spiders are small to medium sized. There are over 30 species with many being shiny black with red markings with the most common being a ventral red hourglass. Yet, there are variations throughout the world. Some regions have white spiders and stripes or dots of varying colors.[1,6] The southern black widow (*Latrodectus hesperus*) and western black widow (*Latrodectus mactans*) are the most clinically significant in North America.[7] Widow spiders are found in every state except

Alaska.[5] The majority of bites occur during warm weather in dark areas around human made objects (boxes, sheds, planters) with more than 75% occurring on extremities.[1,3,4,8] All species produce venom with similar properties and cause a distinctive clinical syndrome.[4]

Widow venom is one of the most potent venoms by volume.[3,8] The venom is produced in a venom gland and injected through fangs. The venom has several components, but the protein alpha-latrotoxin is thought to be toxic to humans.[1,2,4,6] The 120 kDa protein causes release of major neurotransmitters causing depletion of presynaptic vesicles with simultaneous inhibition of neurotransmitter resorption at nerve endings. Acetylcholine, norepinephrine, dopamine, and glutamate are all susceptible.[3-4,9]

The diagnosis of latrodectism is clinical. Being aware of the distribution of spiders is an important factor in identification. The general population can identify widow spiders, which is usually sufficient for treatment.[2] Worldwide there are substantial variations between species. In Australia, a pain syndrome predominates whereas in Europe systemic symptoms are more common.[2,10-12] Even pain patterns vary among different spp.[1] Back and abdominal pain predominate in America.[2,3] Even though clinical effects are usually described as characteristic, it is not uncommon to misdiagnose widow bites.[1]

When envenomation occurs, there is a spectrum in presentation. Usually symptoms begin between 30 to 120 minutes of a high-risk activity and progress over hours.[3,4] Mild envenomation manifests as local skin irritation and localized spasm and pain. Moderate envenomation can cause spasms and pain of the affected extremity and can be accompanied with more generalized pain involving the chest, abdomen, and back. Severe envenomation can cause severe diffuse spasms and pain with more generalized symptoms. Associated symptoms include malaise, headache, fever, hypertension, tremors, convulsions, nausea, vomiting, diaphoresis, and blepharoconjunctivitis (facies latrodectismica). Rarely ileus, priapism, cardiomyopathy, pulmonary edema, hematuria, rhabdomyolysis, toxic epidermal necrolysis, and death occur.[2-6,12,13] The classic "target lesion" a blanched circular patch with a surrounding red perimeter and central punctum within is only seen in 50% of cases. The diaphoresis may have unusual patterns including localized to site of the bite, regional or bilaterally below the knees.[2] Symptoms may wax and wane and may last days to weeks. [3,6,13] In 2014, the National Poison Data System had close to 1,700 reported exposures of which nearly 800 were seen in a healthcare facility; 6.5% had no symptoms, 58% had a minor envenomations, 34% had moderate envenomations, and 1.5% had severe envenomations with no deaths.[14] If labs are checked, there may be an elevation in white blood counts, liver enzymes, glucose, and creatine phosphokinase, as well as hematuria.[3]

Treatment is managed depending on the severity. All envenomations should receive good wound hygiene and receive a tetanus update, if necessary. Heat has been found to worsen pain. Prophylactic antibiotics are not recommended. Mild envenomation are treated with oral analgesics and muscle relaxants. Moderate and severe envenomation may require parenteral analgesics, benzodiazepines, and antiemetics.[2-6,15] Calcium and magnesium, although commonly used, have been ineffective when studied.[2,3,5]

Severe envenomations may require administration of antivenom with the goal of decreasing pain and avoiding hospitalization. In 1926, Los Angeles General Hospital gave 4 envenomed patients intramuscular injections of serum drawn from previously envenomed patients with a good respose.[5] In 1936, Merck licensed a widow antivenom.[15] The current antivenom available in the United States is a partially purified equine-derived IgG antibody, which is administered intravenously.[5] As opposed to Australia where widow antivenom is the most administered antivenom in the continent, in the Unites States, it is in rarely used as there is a limited supply and a reluctance to use because of perceived high risk of allergic reactions.[1,3,5] Originally, antivenom was listed as having an incidence of anaphylaxis between 9% to 80%.[4] Current data suggest that allergic reactions occur in up to 9% of patients, serum sickness in 2-16%, and anaphylaxis in <1%.[2,3,5,10] Data from the National Poison Data System collected between 2000 and 2008 listed 2 cardiac arrests from severe envenomation in approximately 10,000 patients with widow envenomation who had available follow up. Of those patients, 3.78% received antivenom with a 4.5% ADR but no anaphylactic responses or deaths.[16] Antivenom's current indications is pain refractory to opioids and muscle relaxants, life threatening effects, and possibly pregnancy given increased concern for miscarriage.[5]

In general, if antivenom is considered, there should be communication with the poison control center. The most referenced study in American Widows is a retrospective chart review at an urban toxicological referral center that enrolled 163 patients from 1982 to 1990. They categorized patients on severity. 58 patients received antivenom. Of those who received antivenom only 12% were hospitalized as opposed to 52% that did not. One patient died due to severe bronchospasm after antivenom.[3] The author's conclusion "antivenin should be restricted to patients who have severe envenomation and no allergic contraindications and in whom IV or IM analgesics were unsuccessful for pain relief." It is recommended that traditional means should be exhausted prior to using antivenom. Although the manufacturer recommends skin testing, it is unreliable, and many experts believe that it delays treatment and should be abandoned.[6] A highly purified F(ab)2 antibody is undergoing clinical trials. From prior experience with snake antivenoms, they are less immunogenic have a larger volume of distribution but shorter half-lives. This is similar to a formulation used in Mexico.[5]

If the decision is made to administer the antivenom, informed consent should be obtained. It should be administered in a setting where there is advanced airway equipment and ability to manage severe allergic reactions. Premedicating patients has not been found to be helpful.[6]

Figures

Figure 1. A black widow outside Olive View-UCLA Medical Center.

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