Letters to the Editor

Black Widow Spider Envenomation in a 36-Year-Old Man

To the Editor:

We are reporting here a case of local and systemic envenomation resulting from a bite by a black widow spider (*Latrodectus hesperus*). Bites from spiders of this genus (the widow spiders), especially the black widow, have been reported as the leading cause of death from spider envenomation.^{1,2} The female *L. hesperus* has an average body length of 17 mm and a leg spread of 40 mm.³ Only the female is dangerous to humans; the male, a much smaller spider, is incapable of biting through human skin. The effect of widow spider venom has been considered extremely toxic, especially for children.⁴ The venom of *L. hesperus* has a median lethal dose of 1.39 mg/kg in rats.¹ The major component of this venom is a neurotoxin, which stimulates the release of transmitters from nerve terminals, especially the neuromuscular junction, and is followed by blockage of neurotransmission via depletion of synaptic vesicles.⁵

While standing on the ground barefoot, a healthy 36-year-old man (weight, 82 kg) was bitten on the second toe of his left foot by a spider. Because he is a farmer, the patient is familiar with the major types of spiders in this area, and he identified the specimen as a black widow spider (the only widow spider indigenous to this area). He immediately sought medical care at our hospital.

Upon admission, 2 hours after being bitten, he was conscious and found to be in good general condition. Vital signs upon arrival included respirations 24 breaths per minute, blood pressure 120/85 mm Hg, pulse rate 140 beats/min, and rectal temperature 36.5°C. The only symptoms pertaining to the spider bite were a pinpoint puncture wound, mild erythema and paresthesia on the inner aspect of the second left toe, and mild abdominal pain. He was admitted to the general medical ward for observation. Initial treatment consisted only of intravenous fluids. Four hours after envenomation the patient became agitated and restless, and he developed general muscle pain and cramping, especially severe abdominal pain. Physical examination revealed abdomen muscle rigidity, chills, and diaphoresis. His blood pressure rose to 180/95 mm Hg and his pulse quickened to 150 beats/

min. Laboratory analyses including hepatic and renal functions, blood count, coagulation tests, and electrolytes were within the normal range. His hypertension was treated with sublingual nifedipine 10 mg every 4 to 6 hours (total dose 50 mg). In addition, a single dose of 10% calcium gluconate was administered intravenously in an effort to relieve the muscle spasms. However, the pains from the abdominal muscle spasms were only partially relieved; therefore, the patient received morphine hydrochloride 5 mg IV at a rate of 1 mg per minute. The patient required repeat doses of morphine (3 times a day for 48 hours) to keep his pain under control. Blood pressure was reduced with nifedipine treatment, but it did not return to normal until 48 hours postenvenomation. The patient's condition stabilized and systemic symptoms were reduced gradually within 48 hours; therefore, the patient was not treated with antivenom. He was discharged in good condition 3 days after envenomation.

Our patient developed 2 predominant findings: severe abdominal pain with muscle spasms, and hypertension. The venom's action is primarily attributed to the release of acetylcholine at the neuromuscular junction, thereby causing muscle spasm. The release of neurotransmitters from nerve terminals in the brain, sympathetic ganglia, and from some adrenergic terminals induces the hypertension. The treatment consisted mainly of supportive therapy. In more severe cases, treatment with specific antivenom may be required.

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References

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