Use of Antivenin to Treat Priapism After a Black Widow Spider Bite

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ABSTRACT. Black widow spider envenomation (BWSE) is commonly associated with severe abdominal pain, muscle cramping, and hypertension. Treatment is generally supportive, and only those with severe complications and worse symptomatology require interventions. The syndrome of BWSE usually involves a pin-prick sensation at the bite, which is brief in duration. Severity of envenomation depends on the size of the spider, depth of the bite, and the age and size of the victim. Children, the elderly, and persons with cardiovascular disease are considered at high risk for serious complications and worse symptomatology. Approximately 30 to 60 minutes after envenomation, proximal muscle cramping, particularly of the chest, abdomen, and back, occurs. Symptoms can progress to waxing and waning muscle rigidity and severe pain. Findings can often be confused with an acute abdomen, especially in children. Autonomic nervous system stimulation by venom produces nausea, vomiting, sweating, hypertension, and tachycardia. Treatment is generally supportive, and only those

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patients who require repeated doses of medication to relieve symptoms warrant hospital admission.

Treatment is primarily symptomatic, with the use of opiates and benzodiazepines for the management of pain and muscle spasms. Calcium gluconate was previously recommended as the treatment of choice, especially in patients who arrived ≥3 hours after being bitten. However, it was later shown to be ineffective in 96% of patients in a review of 163 cases by Clark et al.

Clark further showed that antivenin was safe and extremely effective in relieving BWSE symptoms. Antivenin is associated with known risks of immediate hypersensitivity and anaphylaxis and can produce serum sickness up to 7 to 10 days after administration. This risk is the same for a half a vial or a full vial, and the dosing of antivenin should be the same in adults and children. Because the goal is neutralization of the venom, the entire vial should be administered. This may have accounted for our patient’s partial resolution until the entire vial of antivenin was given. Woestman et al found that antivenin seemed to bring relief safely to patients in a recent review of 12 children, none of whom developed priapism. Use of BWS antivenin is currently recommended in patients <5 or ≥60 years old with BWSE and respiratory difficulty, marked hypertension, or patient distress not responding to other measures. Antivenin should be administered only in a location where anaphylaxis can be treated, such as an emergency department or intensive care unit.

Priapism is a pathologic condition of penile erection that persists beyond or is unrelated to sexual stimulation. Peak incidence in children is from ages 5 to 10 years and is usually associated with sickle-cell disease or some other hemoglobinopathy. Priapism is thought to occur because of a disturbance in the regulatory mechanisms that maintain penile flaccidity. Priapism can be separated into 2 distinct hemodynamic forms: low flow (ischemic) or high flow (nonischemic). Low-flow priapism results from a decrease in venous outflow from the penis and is characterized by venous stasis and penile ischemia. It is usually a painful, rigid erection characterized clinically by absent cavernous blood flow. Low-flow priapism beyond 4 hours results in a compartment syndrome and requires emergent medical intervention. High-flow priapism results from increased arterial flow into the cavernosal sinuses, which overwhels venous outflow, leading to persistent erection. High-flow priapism is often caused by groin or straddle trauma that results in injury to the internal pudendal artery or its branches. Priapism with BWSE has only rarely been noted. In 1982, Stiles reported treatment with antivenin for presumed BWSE in a 4-year-old with distress, muscle rigidity, hypertension, and priapism. Symptoms were consistent with BWSE, but no spider exposure or puncture wound was documented. The patient had dramatic improvement in his muscle rigidity and discomfort within 30 minutes of antivenin, but priapism and hypertension took >12 hours to resolve. In a case report from Chile, the authors reviewed 89 cases of BWSE and reported only 1 case of priapism. Although latrodectism is relatively uniform throughout the world, some variation in unusual effect might occur, making the incidence of priapism in South America less relevant. Neither the age of the patient nor his specific treatment were given.

One postulated mechanism for priapism in BWSE is through release of neurotransmitters such as acetylcholine and epinephrine, leading to diffuse neuromuscular, autonomic, and central nervous system effects. BMSE may cause overstimulation of the parasympathetic system, resulting in smooth muscle relaxation and increased blood flow into the sinuoids. Priapism also could be caused by the release of acetylcholine at the neuromuscular junction, with resulting obstruction to penile venous outflow by spasm of the ischiocavernous and bulbocavernous muscles.

In our patient, priapism failed to resolve with opiates or benzodiazepines and prompted treatment with antivenin rather than surgical urologic intervention. The patient’s rapid improvement after antivenin suggests that it is effective in treating this aspect of the BWSE syndrome as well as other symptoms.

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