Envenomation by the Scorpion Centruroides exilicauda (C sculpturatus): Severe and Unusual Manifestations

**ABBREVIATIONS.** CPK, creatine phosphokinase; CSF, cerebrospinal fluid; WBC, white blood cells.

Scorpion envenomation continues to be a common occurrence in the southwestern United States. The only medically important scorpion in the United States is *Centruroides exilicauda* (also known as *C sculpturatus*), a scorpion found mainly in Arizona, but also found in parts of Texas, New Mexico, California, and northern Mexico.1

Envenomation by *C exilicauda* is clinically significant because of potent neurotoxins injected from the terminal tail segment of the scorpion into its prey.2 Clinical effects include tachycardia, local pain, restlessness, roving eye movements, hypertension, respiratory distress, tachypnea, hypersalivation, slurred speech, and stridor. These signs and symptoms are usually more severe in children, especially those younger than 5 years of age.3,4 Most envenomations are relatively minor.

Lethal scorpion envenomations are a serious problem in many parts of the world.5–11 The deaths are primarily due to cardiovascular collapse, although respiratory failure and defibrination syndrome have been described.9 These scorpions produce neurotoxins similar to that of *C exilicauda*.12

We describe the clinical course of four children envenomed by *C exilicauda*, each presenting with an unusual or unusually severe manifestation of this event. Two required endotracheal intubation for respiratory failure and metabolic acidosis. Two had marked elevations of creatine phosphokinase (CPK), and one had a severe multiorgan system disease process. Two were noted to have pleocytosis of the cerebrospinal fluid (CSF), a finding not previously reported in the medical literature.

**CASE 1**

A 3-month-old boy was stung on the face by a small scorpion. He soon experienced twitching of his extremities along with copious salivation and inspiratory stridor. The boy was quickly taken to a local emergency room where a diagnosis of respiratory failure secondary to airway obstruction was made. An arterial blood sample obtained as he received supplemental oxygen by face mask had an oxygen tension (PO2) of 92 mm Hg, a carbon dioxide tension (PCO2) of 25 mm Hg, and a pH of 7.12. The trachea was intubated and the patient was transferred to the University Medical Center's pediatric intensive care unit. On admission his temperature was 36.6°C, and heart rate was 174 beats per minute. He had inspiratory stridor and flailing of all extremities. Five hours after admission the boy pulled out the endotracheal tube. He was asymptomatic the following morning and was discharged from the hospital. The offending scorpion was later identified as *C exilicauda*.

**CASE 2**

A 9-month-old girl was well until several hours prior to admission. She abruptly began to cry inconsolably in her bedroom. This was followed by thrashing, arching of the back and head, and two episodes of emesis. There was no history of fever, trauma, or ingestion. On admission her rectal temperature was 39.7°C, and her heart rate was 210 beats per minute. She had thrashing movements of her extremities and roving eye movements, but would make eye contact when her name was called. Analysis of the cerebrospinal fluid revealed a white blood cell (WBC) count of 9/mm3 (92% mononuclear forms), a red blood cell count of 1623/mm³, 74 mg/dL of glucose, and 21 mg/dL of protein. Complete blood cell count was unremarkable and the platelet count was 558,000/mm³. The ammonia, calcium, and electrolyte levels were normal. A screen of the urine for toxic substances was negative. Viral studies were not obtained. Specimens for culture of the blood and CSF were negative for growth.

The patient was managed with intravenous fluid and sedation. Because the physician suggested that this was a typical scorpion envenomation, the patient's mother later searched in the house and found a dead scorpion between the sheets of the child's bed. The scorpion was subsequently identified as *C exilicauda*. Within 24 hours of admission the patient was behaving normally.

**CASE 3**

A 14-month-old Mexican boy woke suddenly in the early morning and screamed loudly and inconsolably. On arrival at the bedside his mother noticed that he was cyanotic, salivating, and agitated. His extremities were flailing wildly. He had a 1-cm red lesion on his side that soon disappeared and a scorpion was noted crawling away from him. He was quickly taken to a local emergency clinic where he was noted to have cyanosis, a “clenched jaw,” copious salivation, and abnormal flexion of his upper extremities.

While at the emergency room in the border town of Nogales, Mexico, the patient received one vial of horse-derived scorpion anti-venom, 5 mg of diazepam, 10 mg of furosemide, and dextrose intravenously and oxygen by
mask. Because of persistent cyanosis and the abnormal mental status, the patient was transferred to a hospital in Nogales, Arizona. There he presented similarly with cyanosis, jaw spasms, and flexion of the upper extremities. Chest roentgenogram revealed bilateral fluffy infiltrates. Analysis of an arterial blood sample obtained while he was receiving mask oxygen revealed Po2 of 80 mm Hg, Pco2 of 35 mm Hg, and pH of 7.10. A diagnosis of respiratory failure was made and the patient was intubated and given positive pressure ventilation. The patient was transferred to University Medical Center for further evaluation.

On admission, he was in circulatory shock with poor peripheral pulses and poor skin perfusion. He was comatose, responding only to painful stimulation with flexion. Serum tests at the time of admission, approximately 12 hours after envenomation, revealed sodium concentration of 139 mEq/L, potassium of 5.6 mEq/L, chloride of 103 mEq/L, total carbon dioxide of 17 mEq/L, blood urea nitrogen concentration of 61 mg/dL, and creatinine concentration of 2.0 mg/dL. The WBC count was 48 000/mm³, the hemoglobin concentration was 10.9 g/dL, and the platelet count was 310 000/mm³. Lumbar puncture was performed and analysis of the CSF revealed a red blood cell count of 0/mm³, a WBC count of 17/mm³ (100% monocytes), a glucose concentration of 24 mg/dL, and a protein concentration of 54 mg/dL. The prothrombin time was 24 seconds, and the partial thromboplastin time was 43.6 seconds. Other blood concentrations included an alanine aminotransferase of 269 U/L, an amylase of 1510 U/L, and an ammonia of 32 mmol/L. All laboratory studies noted above were obtained within 2 hours of admission. Specimens of blood, CSF, and urine were obtained for bacterial culture and the patient was given ampicillin and cefotaxime empirically. Cerebrospinal fluid, nasopharyngeal, and rectal viral cultures were obtained. All bacterial and viral cultures resulted in no growth.

Shortly after admission the patient became more ill with acute hypotension and gross bleeding through the endotracheal tube and nasogastric tube. A serum fibrinogen level at that time was 133 mg/dL and fibrin degradation products were present. The patient was resuscitated vigorously with crystalloid and colloid fluids, as well as cardiotonic agents. The abnormal bleeding subsided approximately 24 hours after admission, and the coagulation profile normalized. Amylase and alanine aminotransferase levels were measured on the third day and were in the normal range. Lumbar puncture was repeated 48 hours after envenomation, and analysis of the CSF revealed a WBC count of 1/mm³, a glucose concentration of 80 mg/dL, and a protein concentration of 27 mg/dL.

On the day of admission the serum CPK level was 10 600 U/L with 0.7% myocardial band fraction. An electrocardiogram was normal. Cardiac function (as judged by the need for inotropic support) improved by the seventh day.

The patient experienced non-oliguric renal failure, requiring 6 days of peritoneal dialysis. Results of renal function and electrolyte studies ultimately returned to normal. Despite invasive monitoring and effective management of mild intracranial hypertension, his neurological status improved only moderately during the next week. He had spastic quadripareisis and significant neurological dysfunction on discharge.

CASE 4

A 3½ year old boy who was stung by a yellow scorpion had immediate local pain, coughing, and wheezing. He then began uncontrollable "flailing" of his arms and legs. On arrival in the pediatric intensive care unit, he was confused, combative, and had roving movements of his eyes and flailing movements of both arms and legs. His rectal temperature was 100.4°F. Oxygen saturation by pulse oximetry was 90%. A complete blood cell count showed a WBC count of 22 000/mm³ and hemoglobin concentration of 12.0 g/dL. The differential WBC count showed a slight shift to the left. The platelet count was 342 000/mm³. Serum chemistry studies were unremarkable except for a serum CPK level of 2191 U/L.

Supplemental oxygen and sedative medications were provided for several hours. Six hours after admission a CPK level was 4995 U/L. Serial analyses of the urine were negative for myoglobin. All clinical signs and symptoms resolved within 24 hours, and he was discharged to home.

DISCUSSION

The scorpion C exilicauda is the only medically important scorpion in the United States.1-3 C exilicauda is found mainly throughout the state of Arizona and in parts of California, Texas, Nevada, New Mexico, and northern Mexico. It dwells among trees and is considered a climbing scorpion.2

C exilicauda and other scorpions generally attack humans only as a gesture of self-defense. The sting is accomplished by grasping onto the prey with its pincers, followed by thrusting of the tail over the body, penetrating the victim's flesh.5

Mild envenomation produces immediate local pain and paresthesias, often followed by perioral numbness. More significant envenomations lead to muscle jerking, tachycardia, restlessness, hyperpyrexia, "roving eye movements," hypertension, blurred vision, and elevated peripheral WBC count.2,3

Signs and symptoms involving the respiratory system include tachypnea, excessive salivation, stridor, and wheezing.2,3 Rimsza et al3 reviewed 24 cases of scorpion envenomation in Phoenix, Arizona, from 1970 through 1978. Six had respiratory distress—four with stridor, two with expiratory wheezing. Two required assisted ventilation, both after receiving 15 mg/kg of phenobarbital.

It appears that the severity of symptoms following envenomation is inversely related to the age of the victim. Likes et al4 reviewed more than 1000 cases of scorpion envenomation between 1980 and
1981 that presented via telephone calls to the Arizona Poison and Drug Information Center. No deaths occurred in the series. However, children younger than 2 years of age often were agitated or hyperexcitable and frequently required hospital admission. Curry et al. reviewed 673 scorpion stings in Phoenix. Only 52 patients had any systemic signs or symptoms. Sixteen of their 20 patients with severe envenomations were younger than 10 years old.

_C. exilicauda_ was the leading cause of death by venomous animals in Arizona during the 1930s and 1940s. The rate of death from scorpion envenomation dropped dramatically from 1940 to 1970. This drop was attributed to good scorpion control and eradication and improved therapeutic practice. There have been no reported deaths following scorpion envenomation in Arizona since 1968, suggesting that envenomation by _C. exilicauda_ is not as dangerous as previously believed.

In other parts of the world, envenomations by certain scorpions that produce clinical syndromes similar to that of _C. exilicauda_ cause many deaths. _Buthus quinquestriatus_, a yellow scorpion found in Israel and North Africa, has been shown to cause myocardial damage and acute cardiovascular collapse. A 1960s series of hospitalized Israeli scorpion sting victims revealed a 15% mortality rate; a recent series from Israel showed a 4% mortality rate. _Buthus tamulus_, found in India, has been shown to cause myocarditis, pulmonary edema, and disseminated intravascular coagulation. In vitro, _C. exilicauda_ and _B quinquestriatus_ venoms have similar effects resulting in activation of neuronal sodium channels with subsequent excessive firing of neurons. Also, these venoms increase blood pressure and plasma renin levels in rats to a similar extent.

In two of our patients there was clinical and laboratory evidence of respiratory failure following envenomation from _C. exilicauda_. Both had severe upper airway obstruction, and one had pulmonary edema. As previously noted, stridor, hypersalivation, and tachypnea are recognized consequences of _C. exilicauda_ envenomation. However, respiratory failure secondary to upper airway obstruction or pulmonary edema is unusual.

One of our patients had an amylase level of 1510 U/L (normal <200 U/L). In Trinidad, the most common cause of pancreatitis is envenomation by the scorpion _Tityus trinitatis_. Venoms from other _Tityus_ species and _B quinquestriatus_ have been shown to create pancreatic damage in dogs. Perhaps envenomation by _C. exilicauda_ also causes pancreatitis.

Two of our patients were noted to have evidence of rhabdomyolysis acutely following envenomation. To our knowledge, elevated levels of serum CPK have been documented in only one other child following envenomation by _C. exilicauda_ (GR Bond, MD. Verbal communication. May 1990) Further investigation of rhabdomyolysis and possible myocardial involvement is warranted.

Two of the four cases were noted to have sterile pleocytosis of the CSF. Lumbar puncture was performed in both because of lack of "history of scorpion sting" at the time of presentation. In both cases the clinicians initially believed that CSF pleocytosis suggested viral meningencephalitis and was not consistent with scorpion envenomation. However, the diagnosis of scorpion envenomation became apparent after detailed review of the history and clinical findings. Lumbar puncture is rarely performed in patients following scorpion envenomation. Therefore, these two patients may have exhibited a not uncommon consequence of scorpion envenomation. The clinical significance of this is more difficult to determine. Case 2 appeared to have normal neurological function on the next day, whereas case 3 left the hospital 30 days later with severe neurological impairment. Nevertheless, scorpion envenomation must be considered in the differential diagnosis of CSF pleocytosis.

**CONCLUSIONS**

Envenomation by _C. exilicauda_ may not be as benign as recently thought. Two of our patients showed clinical signs of respiratory failure requiring tracheal intubation, one with multisystem failure. More data are necessary to determine the significance of CSF pleocytosis and elevated serum CPK, each seen in two of our patients. Perhaps _C. exilicauda_ is dangerous to some individuals, much like the scorpions in the Middle East, Far East, and Latin America, but our public health standards result in a lower incidence of envenomations and therefore the rarity of uncommon severe complications such as respiratory failure, cardiac failure, disseminated intravascular coagulopathy, and pancreatitis.

**ADDENDUM**

Since this paper was submitted for publication we have had another patient present with severe multiorgan system involvement from a scorpion envenomation, much like case 3. The 14-month-old boy presented with acute upper airway obstruction, pulmonary edema, and profound mixed metabolic and respiratory acidosis (arterial pH 6.96 and PCO₂ 58), as well as the typical neurological manifestations. Within 2 hours of _C. exilicauda_ envenomation, endotracheal intubation and mechanical ventilation were provided. Chest roentgenogram revealed pul-
monary edema. Prothrombin time and partial thromboplastin times were mildly prolonged at 16.3 and 35.7 seconds. Serum creatine kinase level was 5140 IU/L, and serum amylase level was 902 IU/L. His laboratory and clinical status returned to normal within 3 days.

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REFERENCES

Antiphospholipid Antibodies and Stroke in an Infant

ABBRVIATIONS. APA, antiphospholipid antibodies; aPTT, activated partial thromboplastin time.

Cerebral infarctions are rare in children but have been associated with a variety of conditions including congenital heart disease, migraines, blood dyscrasias, homocystinuria, and infections. Inasmuch as some predisposing conditions are associated with recurrence of infarction and other complications, an extensive evaluation is required to determine the cause of the infarction and any possible treatment.

“Lupus anticoagulants” and “anticardiolipin antibodies” are different types of antiphospholipid antibodies (APA). Over the past decade, an association has been demonstrated between APA and ischemic events in adults.1−3 Antiphospholipid antibodies have also been found among adolescent patients with and without other autoimmune diseases. Ischemic complications have occurred in some of these patients.4,5

We present a case of cerebral infarction associated with APA in an 8-month-old child. This is the youngest reported patient with cerebral infarction associated with APA.

CASE REPORT

The male patient was noted to have dolichocephaly at 4 months of age. Skull radiographs showed sagittal synostosis, and surgical repair was performed at 7 months. No coagulation tests were performed at the time of the surgery. Intraoperatively, 200 mL of blood was lost, and 100 mL of packed red blood cells was transfused. The postoperative course was unremarkable.

Four weeks after surgery, the patient fell down five steps without loss of consciousness. Examination revealed swelling along the sagittal suture line; neurological examination results were normal. Computed tomographic scan of the head revealed a subgaleal hematoma but no intracranial abnormalities. The fluid accumulation became more tense over 24 hours and was tapped with