Alerts, Notices, and Case Reports

Ingestion of Poison Hemlock (*Conium maculatum*)

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DESPITE ITS PROFOUND toxicity and wide distribution, poison hemlock (*Conium maculatum*) is an uncommon cause of poisoning in children. We report a case of poison hemlock ingestion.

Report of a Case

The patient, a healthy 4-year-old boy, and his father ingested the green tops of "wild carrots" growing in their backyard at about 3 PM on the day of admission. Within 30 minutes, the child became sleepy and took a nap. This was unusual for him. Two hours later, his father could not wake him up, and the patient had vomited green material in his bed. He was immediately taken to an emergency department. The child had no history of trauma, seizures, or fever. His father remained asymptomatic.

On physical examination, the patient's pulse rate was 100 beats per minute, and the respiratory rate was 26 per minute. His pupils were small and reactive, and the gaze was disconjugate. His neck was supple. The lungs, heart, and abdomen were unremarkable. There were no signs of trauma. On neurologic examination, there was semipurposeful response to noxious stimuli in the upper extremities and withdrawal in the lower extremities.

A complete blood count, electrolytes, blood urea nitrogen concentration, serum creatinine level, glucose level, prothrombin and partial thromboplastin times, and arterial blood gas values were within normal limits. A toxicology screen was negative. Radiographs of the chest and abdomen were normal.

Infusions of a dextrose solution and nalorphine hydrochloride produced no clinical improvement. The patient's stomach was lavaged, and he was given activated charcoal. During the first 90 minutes, he became progressively less responsive, but maintained an adequate gag reflex. Over the next hour, he began responding to simple commands, and within four hours he was awake and talking to his parents. He was discharged two days later with normal physical findings.

Figure 1.—The photograph shows the leaves and stalk of the hemlock plant (*Conium maculatum*).

A plant identified by the patient's father as identical to the others that both he and the patient ate was submitted to the Intermountain Herbarium, Logan, Utah, for taxonomic identification. Analysis by gas chromatography and mass spectrometry confirmed the presence of piperidine alkaloids characteristic of poison hemlock. The leaves contained 850 μg of γ-coniceine per gram of fresh plant.

Discussion

Conium maculatum is a poisonous biennial herb that grows erect to an average height of 1 to 3 m (3¼ to 9¾ ft).[1] The larger stems of maturing plants contain numerous purple spots that are an identifying characteristic. First-year-growth plants have fine, light-green, fernlike leaves (Figure 1) and usually grow no taller than 46 cm (18 in). Poison hemlock has a long white taproot that is solid and parsniplike. Plants generally persist in localized stands because the seeds drop near the parent plant. Occasionally seeds are spread by water, birds, or rodents.

Poison hemlock was introduced into the United States from Europe as an ornamental plant. It has become widespread and frequently grows in waste places, along roadsides, ditch banks, fence rows, and in uncultivated areas or anywhere adequate moisture is available. Its distribution is nationwide.

The toxins in poison hemlock are simple piperidine alkaloids. Coniine and γ-coniceine (Figure 2) are the predominant toxicants and not only have been implicated in overt toxicity in animals and humans but have been shown to induce congenital birth defects in livestock species.[2] The oral median lethal dose in mice for

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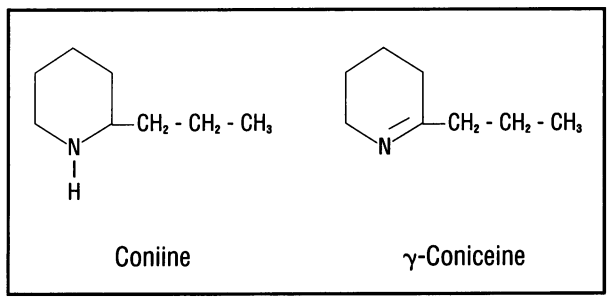

Figure 2.—The graphic formula of the alkaloids coniine and γ-coniceine, the predominant toxins of the hemlock plant, is shown.

γ-coniceine, the most toxic and most plentiful of the alkaloids, is 12 mg per kg.[3]

The mechanism of action of these alkaloids is twofold. The most serious effect occurs at the neuromuscular junction where they act as nondepolarizing blockers, similar to curare.[3] Death, when it occurs, is usually caused by respiratory failure. As a result of their action at the autonomic ganglia, the toxins produce biphasic nicotinic effects, including salivation, mydriasis, and tachycardia followed by bradycardia.[3] Less commonly, rhabdomyolysis and acute tubular necrosis have occurred.[4]

Poison hemlock is often confused with water hemlock (*Cicuta* species) because the two are similar in appearance and belong to the same family. The toxin in water hemlock, cicutoxin, has primarily central nervous system effects, including seizures.

Poison hemlock poisoning may be suspected in patients with an altered level of consciousness, myalgias, fasciculations, or flaccid paralysis following the ingestion of a plant substance. Supportive laboratory data include elevated muscle enzyme levels and myoglobinuria.[5] Elevated values on liver function tests have also been seen.[5] Routine plant toxin screens in biologic specimens are not commonly done except for those substances abused and for which drug screens are available. A method for a multiresidue chemical screen for alkaloids in plant material was recently described that provides the basis for confirming a case of poison hemlock or water hemlock toxicosis.[6]

Because no antidote exists for coniine poisoning, treatment is supportive.[7] Respiratory support and gastric decontamination should be instituted immediately. Anticonvulsants should be administered as needed. Forced diuresis may be useful in preventing renal failure from rhabdomyolysis and myoglobinuria.

The relatively short course and limited symptoms that our patient manifested can probably be explained by the small amount of plant ingested, the vomiting at home, and finally, the gastrointestinal decontamination that limited the quantity of toxin absorbed.

That ingestions of poison hemlock are not more common has been attributed to the plant's "mousy" odor, bitter taste, and burning of the mouth, throat, and abdomen on ingestion.[8] The father of this patient said that the plants tasted like "carrot tops."

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Multiple Organ Dysfunction Caused by Parvovirus B19

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HUMAN PARVOVIRUS B19 infection was first described in 1975. Erythema infectiosum (fifth disease) is the most commonly recognized parvovirus B19 infection in children. It has also been shown to be the primary etiologic agent of aplastic crisis in persons with chronic hemolytic anemia as well as being associated with cases of arthritis, purpura, and congenital infections manifesting as spontaneous abortion, stillbirth, or fetal hydrops.[1,2] We describe the case of a child with multiple organ dysfunction involving the skin, liver, heart, and hematopoietic system caused by parvovirus B19 infection, and we review published literature on B19-induced myocarditis and multiple organ dysfunction.

Report of a Case

The patient, a 19-month-old previously healthy female infant presented with an eight-day history of fever and a generalized erythematous macular rash that was first noted on the abdomen. Fifteen days before admission, she was admitted to a hospital with two generalized tonic-clonic seizures, bloody diarrhea, vomiting, and fever.

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