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CASE REPORT

A Case Report of Water Hemlock Poisoning

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Introduction: Water hemlock poisoning is an uncommon cause of seizures, gastrointestinal upset, and renal failure. This poisoning occurs infrequently and is likely to go unrecognized without a proper history and consideration in the differential diagnosis.

Clinical findings: A 23-year-old male with an unremarkable past medical history presented to the emergency department after being found unresponsive at a farm where he was employed. He had several tonic-clonic seizures en route. Initial evaluation was unremarkable and included toxicology screening, lumbar puncture, and brain imaging.

Diagnoses, Interventions, and Outcomes: The patient was treated with broad-spectrum antibiotics and antivirals with a suspected diagnosis of viral encephalitis. Over the next several days of hospitalization, he developed severe rhabdomyolysis and renal failure, and dialysis was anticipated. Upon further investigation, it was discovered that on the morning of his presentation, the patient made himself tea with a plant he had dug up while fly fishing. He believed the plant was valerian root after researching it on the Internet. The plant was later identified as water hemlock. With supportive care, the patient’s mentation cleared, and his renal failure spontaneously resolved without the need for dialysis. His symptoms fully resolved, and he was discharged home.

Conclusions: This case illustrates an unusual etiology of seizures and rhabdomyolysis and the need for careful history taking. The interest in nontraditional medicine and the ease of finding amateur foraging data on the internet have greatly raised the possibility of accidental toxic ingestions.

Keywords: water hemlock, toxicology, accidental ingestion, rhabdomyolysis
patient’s vehicle were identified by an expert as *Cicuta maculate*, which was confirmed after a visit to the site where the roots were collected (Figure 1).

**DISCUSSION**

Plants in the *Cicuta* genus are historically considered the most poisonous native plants in North America, based on 30% case-fatality data (1900–1975: 78 exposures with 28 deaths; 1979–1988: 5 known deaths; 1988–present: at least 2 known deaths, one of which occurred in Maine).² Cicuta is commonly known as water hemlock but has other common names, including beaver poison, spotted cowbane, and poison parsley.² It is a shrub that grows up to 2.5 meters high in wetlands, along streams, and in marshes throughout North America and Europe. Most exposures to *Cicuta* occur because the plant is misidentified as wild parsnip, turnip, carrot, or ginseng.³ Although all parts of the plant contain toxin, consumption of the root correlates with seizures.⁴

*Cicuta* can also be confused with *Conium maculatum*, the historically famous poison hemlock consumed by Socrates. While *Cicuta* and *Conium* both have umbrella-shaped collections of white flowers and fern-like leaves, poison hemlock has smooth purple-spotted stems, a branched root system, and a foul odor.⁵ These plants may initially cause similar nicotinic symptoms of toxicity (seizures).

Water hemlock is a rare ingestion. Only two cases have been reported in the medical literature over the last 20 years.²,⁶ A review of the literature described the typical rapid onset of gastrointestinal (e.g., nausea, vomiting) and neurological (e.g., confusion, seizure) symptoms within 30 minutes of consumption.⁴

The principle toxin in water hemlock is cicutoxin, a 17-carbon diacetylenic diol that acts as a non-competitive antagonist of Gamma-Aminobutyric acid (GABA A) receptors. Through this mechanism, cicutoxin blocks disinhibition of neuronal depolarization to promote uncontrolled signal propagation.⁴ Death follows acutely from respiratory failure as a result of prolonged seizures. Treatment of patients with status epilepticus depends on the aggressive early cessation of seizure activity to reduce the incidence of complications. Generous use of benzodiazepines, barbiturates, and propofol can reduce morbidity.⁷,⁸ Frequently described complications include rhabdomyolysis, myoglobinuria, hyperthermia, lactic acidosis, and renal failure.⁹ The rise in creatinine phosphokinase is often greater than expected from the seizure duration, suggesting a direct myotoxic effect.⁴ Renal failure was a late complication in this case, and the apparent glomerular filtration rate declined after most neurological symptoms subsided.

In the era of easily-accessible plant photos and increased interest in non-traditional medicines, the risk of plant misidentification is on the rise. Unintentional toxic ingestion must be considered in similar patients with a very abrupt onset of symptoms. It is also important to educate the public about the risks involved with inexperienced foraging.

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**Figure 1.** Leaves removed from patient’s truck (left) and a mature rhizome (right) taken from the riverbank at the foraging site. These samples were later identified as *Cicuta maculata* by a botanist at the University of Maine. Photos by author used with permission.

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