CASE REPORT

Intravenous Poison Hemlock Injection Resulting in Prolonged Respiratory Failure and Encephalopathy

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Abstract

Background Poison hemlock (*Conium maculatum*) is a common plant with a significant toxicity. Data on this toxicity is sparse as there have been few case reports and never a documented poisoning after intravenous injection.

Objectives We present a case of intravenous poison hemlock injection encountered in the emergency department.

Case Report We describe a 30-year-old male who presented to the emergency department after a brief cardiac arrest after injecting poison hemlock. The patient had return of spontaneous circulation in the emergency department but had prolonged muscular weakness and encephalopathy later requiring tracheostomy.

Conclusion Intravenous injection of poison hemlock alkaloids can result in significant toxicity, including cardiopulmonary arrest, prolonged weakness, and encephalopathy.

Keywords Hemlock · Injection · Poison hemlock · Encephalopathy

Introduction

Poison hemlock (*Conium maculatum*) is an herb in the Apiaceae family located in all continents except Antarctica. It is commonly found in damp regions, such as riverbanks, small streams, and roadside ditches. Due to its appearance, location, and name, it is commonly confused with water hemlock (*Cicuta* species). It causes toxicity through nicotinic alkaloids, the most potent being coniine [1]. Due to the widespread distribution of this plant, and its visual similarities with other edible plants, there have been cases of oral ingestion of poison hemlock, resulting in respiratory failure due to muscular weakness resulting in failure to ventilate. To our knowledge, there have been no documented instances of intravenous injection of poison hemlock or its alkaloids to date.

Case Report

A 30-year-old male with a history of schizoaffective disorder presented to the emergency department after suffering cardiopulmonary arrest. On initial presentation, there was no information known about the patient other than what was left in a suicide note found on him by paramedics. Prior chart review revealed a history of non-compliance with medications, and no other past medical history. Prior to the arrest, the patient had notified an acquaintance, who subsequently called EMS; however it is unknown the time from the call, to the time of arrest. Paramedics found him unresponsive and pulseless, and initiated cardiopulmonary resuscitation (CPR). He was in pulseless electrical activity, and after less than 10 min of advanced cardiac life support, including chest compressions, mechanical ventilation through a supraglottic airway device, and administration of bicarbonate and epinephrine intravenously, he had return of

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spontaneous circulation with a sinus tachycardia rhythm. Paramedics did not report any seizure-like activity.

On arrival to the emergency department, he was unable to give information due to his condition, but a suicide note found at the scene stated he injected poison hemlock, and he had fresh track marks on his left arm. Additionally, there was an unidentified liquid solution in a syringe recovered from the scene. This was presumed to be poison hemlock. This liquid was found to contain coniine, N-methylconiine, and conhydrine, the main alkaloids in poison hemlock, via high performance liquid chromatography with mass spectroscopy.

The patient's initial vital signs were a heart rate of 134 bpm, blood pressure of 110/87 mmHg, temperature of 35.3 °C, with respiratory rate controlled at 24 breaths per minute through mechanical ventilation with 99% oxygen saturation. Physical exam showed a young male with 4-mm pupils that were fixed. He had no diaphoresis and had a normal amount of oral secretions. He had absent movement, respiratory effort, and no gag or cough reflexes. There were no signs of oral or tongue trauma. There was no bladder or bowel incontinence. He had not received any sedation and had no change in neurological status while in the emergency department. Complete blood count, comprehensive metabolic panel, and creatinine kinase were all within normal limits. A six-panel urine drug screen, which qualitatively measures for barbiturates, amphetamine, cocaine, marijuana, benzodiazepines, and opiates, was negative. Serum salicylate, acetaminophen, and ethanol concentrations were negative. He had an elevated lactic acid of 4.6 mmol/L, which normalized after intravenous fluid hydration during his first day after admission. A non-contrast head CT showed no acute intracranial abnormality, and electrocardiogram revealed sinus tachycardia with no other abnormalities.

Despite a normal blood pressure on presentation, he developed hypotension with a blood pressure of 71/45 mmHg shortly after endotracheal intubation. He was temporarily on norepinephrine infusion with a maximum of 5 mcg/kg/min for hypotension in the emergency department, and this was weaned off within 12 h of admission without recurrence of hypotension. His hospital course was not complicated by rhabdomyolysis or acute kidney injury. Other than the lactic acidosis on presentation, which resolved over the first hospital day, there were no significant laboratory abnormalities on the remainder of his hospitalization. He had profound weakness that slowly improved over his first week of hospitalization. He was able to regain an adequate respiratory effort and pass a spontaneous breathing trial 4 days after admission. However, he had significant agitation, and he was not able to be extubated at this time. An MRI of his brain was performed on his 4th day of admission, which was negative for infarction, anoxic injury, or other acute abnormality, as read by our staff neuroradiologists. Due to persistent agitation, this was repeated along with an MRI of cervical spine on Day 11 of hospitalization. The MRI brain was again interpreted by

neuroradiology as negative for acute abnormality, including anoxia, and the MRI of cervical spine was interpreted by neuroradiology as negative for acute cord injury. Electroencephalograms on days 1, 2, 7, and 11 were consistent with moderate to severe encephalopathy and without evidence of seizure. Because of persistent agitation accompanying his encephalopathy, extubation was not possible and a tracheostomy was performed on hospital day 10 and a feeding tube on hospital day 11. He had a very gradual improvement until discharge to a rehabilitation facility after 23 days of hospitalization. On discharge, his encephalopathy had improved to being able to follow simple commands, and he had recovered most of his strength. After an extended stay in rehabilitation, the patient remained with some mild cognitive deficits and hypersexual behavior, but greatly improved since discharge.

Discussion

Poison hemlock causes toxicity through nicotinic alkaloids. It contains eight piperidine alkaloids, the most potent being coniine, with the other main toxic contributors being gamma-coniceine and N-methylconiine. The concentration of alkaloids found in the plant is highly variable. Factors influencing this include the age of the plant, season, and precipitation. Toxicity from foraging is well-described in livestock. Symptoms observed in cattle, pigs, and sheep include a biphasic presentation. Initially, they will suffer from trembling, excessive salivation, ataxia, mydriasis, followed by weakness, bradycardia, slow movements, and eventually coma and death [2]. This is due to an initial stimulation, followed by a dose-dependent blockade of spinal reflexes through their action on the medulla. As a result, there will be early nicotinic effects, followed by a profound indirect neuromuscular blockade [3]. One murine animal study that investigated the effects of intravenous coniine versus oral coniine demonstrated a lower lethal dose and more rapid death when administered intravenously. In these mice convulsions, rigidity and hypertonic limbs preceded death [4].

Although there are few documented cases in literature, human toxicity from poison hemlock has historical significance and been known from antiquity. It causes death by respiratory failure via paralysis from neuromuscular blockade. The most famous description is that of Socrates, who died in 399 BC when forced to drink poison hemlock as a means of execution. Plato wrote an account where he describes Socrates as drinking the poison, initially walking until he developed progressive weakness, resulting in collapse followed by respiratory failure and subsequent death [5]. There have been previously documented cases of poison hemlock ingestion in contemporary medical case reports, however there were

no case reports of intravenous injection found in our literature search. A case report from Hong Kong in 2011 describes a 59-year-old male who ingested the plant. He suffered respiratory paralysis and required mechanical ventilation. He improved within 2 days and was discharged with full recovery [6]. Another case report from 2013 describes a 21-year-old patient who took a mixture of poison hemlock, amitriptyline, and diazepam. This patient had prolonged ventilatory requirements, requiring 14 days of mechanical ventilation until extubation, and 28 days until he had full neurologic recovery. [7] The largest case series is an Italian series from 1991 detailing 17 patients from 1972 to 1990 with hemlock toxicity from eating poisoned songbirds. All experienced neuromuscular toxicity, with four deaths from a combination of renal failure from rhabdomyolysis and prolonged paralysis [8]. There was a pediatric case of a 2-year-old boy who ingested poison hemlock who suffered rapidly progressive muscular weakness necessitating mechanical ventilation. He recovered within 24 h with no lasting effects [9]. This is a small subset of cases of poison hemlock toxicity, but all cases document severe respiratory paralysis of varying duration. There was an absence of muscarinic findings in the case reports reviewed above.

There have been reports of delayed neurological sequelae in poison hemlock ingestion, but with eventual return to neurological baseline in surviving patients. None of the previous case reports have described a persisting encephalopathy as seen in our case. This could be for a number of reasons. It is possible that in previous cases of poison hemlock ingestion, victims would have developed a similar encephalopathy, but they expired before it had developed. The only previous documented intravenous hemlock exposure was in mice where coniine, N-methylconiine, and gamma-coniceine were injected into mice with a significantly lower LD50 than in oral toxicity. It is possible that occult anoxic brain injury could be responsible, however, this would be unusual given his multiple negative MRIs, as well as no evidence of seizure activity. This seems unlikely given his extensive workup but it should be left open for consideration.

A limitation to this case report is the lack of serum or urine assay for coniine alkaloids. However, a syringe was found next to the patient that contained coniine alkaloids, fresh track marks were noted on his left arm, and a note found with the patient stated he had injected poison hemlock.

The case above illustrates that clinical treatment of poison hemlock intoxication is supportive, with focus on mechanical ventilation as poison hemlock causes profound neuromuscular weakness. General management includes consideration of activated charcoal in the cases of oral exposure. In this case, it was not considered as patient was already symptomatic and had a note stating that he injected, rather than ingested poison hemlock [10].

Conclusion

We present a case of intravenous poison hemlock injection in a human. The events preceding this patient's cardiac arrest were not witnessed. Previous cases of poisoning by ingestion describe paralysis with respiratory failure requiring ventilatory support. In one animal model investigating intravenous coniine seizures preceded death. We can only speculate on the events leading to this patient's arrest. He suffered prolonged encephalopathy with agitation and only partial recovery by hospital discharge 3 weeks later. No changes were noted on multiple brain MRIs, and no seizure activity was witnessed or detected on EEG. The management in this case was supportive.

Compliance with Ethical Standards

Conflicts of Interest The authors declare that they have no conflicts of interests.

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