Jimson Weed Poisoning—A Case Report

By Kit Chan, MD

Abstract

Jimson weed, a plant best known among adolescents and young adults for its hallucinogenic properties, grows as a wild herb in the United States. Ingestion of jimson weed produces the toxidrome of anticholinergic intoxication. Understanding and recognizing the classic signs and symptoms of anticholinergic intoxication can help clinicians evaluate persons presenting with jimson weed poisoning.

Introduction

Ingestion of jimson weed *(Datura stramonium)* is fairly common and can lead to intoxication and to anticholinergic manifestations that are potentially dangerous.[1] The plant is a wild herb that grows throughout the United States, usually matures between May and September, is accessible to almost anyone, and is particularly popular among adolescents curious about the plant's hallucinogenic effects. Understanding the signs and symptoms of jimson weed toxicity can lead to early diagnosis and proper case management. Anticipatory counseling for teenagers and parents may also prevent experimentation and resultant harm.

Case Report

The mother of a 15-year-old boy brought him to the emergency department (ED) because of his bizarre behavior, including hallucinating. The mother had been advised by a neighbor that several neighborhood youths had been taken to nearby hospitals after ingesting wild flowers and then hallucinating. The patient's mother had entered the patient's room and found him shaking, mumbling, and trying to pick at nonexistent items. She noted several white flowers in his room and brought them to the ED.

In the ED, the patient was restless, pacing incessantly, and shaking. He was awake, alert, and oriented to name but not to place or time. Vital signs included oral temperature 99.3°F (37.4°C), blood pressure 117/72 mmHg, heart rate 103 beats/min, and respiratory rate 24 breaths/min. Pupils were dilated to 8 mm, symmetric, and minimally reactive to light. Mucous membranes were dry, and bowel sounds were decreased. The extremities were warm to the touch but were not hot. Neurologic examination showed that the patient was confused and mumbling, cranial nerves were intact, and both motor strength and reflexes were within normal limits. During the examination, the patient reached into the air as if trying to catch a nonexistent object.

Results of an emergent fingerstick blood glucose test, complete blood count, chemistry panel, and urinalysis were normal. Results of a toxicology screen were negative for alcohol, benzodiazepines, amphetamines, marijuana, tricyclic antidepressant agents, opiate agents, and phencyclidine. An electrocardiogram showed sinus tachycardia without other abnormality. Cranial structures appeared normal on computed tomography scans administered without contrast medium.

On the basis of both the clinical presentation and a history of ingesting a wild plant, the ED physician suspected jimson weed intoxication, which was confirmed by comparing the mother's plant specimen with a picture of jimson weed (obtained from the Internet). The patient denied any drug use but stated that his friends had given him a blended drink consisting of strawberries, a wild plant, and a small amount of alcohol.

In the ED, the patient received several doses of lorazepam intravenously as treatment for agitation. He was admitted to the hospital for observation and for monitoring. The patient remained stable, and his mental status improved. At a subsequent interview, the patient admitted that he and his friends had consumed jimson weed deliberately: They had tried it for the first time after hearing that it was hallucinogenic. After 36 hours of observation, the patient was discharged from the hospital.

Discussion

Jimson weed is a member of the nightshade family. An earlier name for the plant was Jamestown weed, coined after intoxication from the plant was first recorded in Jamestown, Virginia, in 1676; the name was subsequently shortened to jimsonweed .[2] The same plant is known also as thorn apple, angel's trumpet, stinkweed, and green dragon.[1,2] The plant has been used for centuries to treat asthma, diarrhea, intestinal cramps, and nocturia because of its anticholinergic effects, and its hallucinogenic effects were mentioned in Homer's tale, *The Odyssey*.[3,4]

Jimson weed reaches a height of five feet and consists of large, jagged leaves and trumpet-shaped flowers, that may be white or purple. At maturity, the plant bears green fruit, each containing four compartments and holding as many as 100 seeds.[1,5] Although all parts of the plant

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are poisonous, the leaves and seeds contain the highest concentration of atropine, hyoscyamine, and scopolamine.[6] One hundred seeds contain approximately 6 mg of atropine.[2,5] A dose of atropine exceeding 10 mg is regarded as potentially lethal.[2]

Today, jimson weed poisoning is found primarily among adolescents who seek the hallucinogenic effects of the plant.[7] In 1998, 152 cases of jimson weed poisoning were reported nationally to the American Association of Poison Control Centers, but the true number of cases is undoubtedly far higher.[1]

The anticholinergic effects of jimson weed are attributed to the atropine, hyoscyamine, and scopolamine components. Symptoms of jimson weed toxicity usually occur within 30 to 60 minutes after ingestion. Initial symptoms include hallucinations, dry mucous membranes, thirst, dilated pupils, blurred vision, and difficulty speaking and swallowing.[2] Subsequent effects may include tachycardia, urinary retention, and ileus. Rarely, late symptoms may include hyperthermia, respiratory arrest, and episodes of seizure.[6] Slowing of gastrointestinal motility may prolong elimination of the toxin, thus causing symptoms to persist for 24 to 48 hours.

Classic anticholinergic symptoms include mydriasis; dry, flushed skin; hallucinations; agitation; hyperthermia; urinary retention; delayed intestinal motility; tachycardia; and episodes of seizure.[3,5,7,8] The mnemonic for anticholinergic symptoms—"blind as a bat, dry as a bone, red as a beet, mad as a hatter, and hot as a hare"—thus applies well to jimson weed poisoning.

Effective treatment of jimson weed poisoning requires a primary survey, clinical evaluation and recognition, elimination of the poison, supportive treatment, and continuing observation.[8] The primary survey includes assessment of the ABCs—ie, airway, breathing, and circulation. Although rare, some patients with jimson weed intoxication may be seen for episodes of seizure or coma. If compromise of the airway is suspected, prompt intubation and mechanical ventilation are indicated.

A detailed history and physical examination results obtained after the patient's condition is initially stabilized can often give clues leading to diagnosis of anticholinergic toxidrome, even if jimson weed poisoning is not immediately identified. A common presenting complaint is altered mental status. The patient may have visual hallucinations, auditory hallucinations, or both.[9] Physical examination may show tachycardia and elevated blood pressure. Hyperpyrexia is seen in about 20% of the cases.[3] Other manifestations include mydriasis, blurred vision, decreased bowel sounds, and dry mucous membranes.

A toxicology screen is useful to rule out concomitant use of other drugs. Most documented lethal cases of jimson weed ingestion occur in persons with polysubstance abuse, including use of jimson weed combined with alcohol, marijuana, or cocaine.[7] Drug screens usually do not detect pure anticholinergic poisons, and other laboratory tests are usually not helpful for identifying jimson weed as the cause of symptoms.[3]

Absorption of jimson weed may be minimized either by using an agent that binds to the toxins or through removal of gastric contents by inducing emesis or administering gastric lavage. Activated charcoal binds to the toxins in jimson weed and decreases overall absorption of these toxins.[5] The usual oral dose of activated charcoal for adults is 1 g/kg. If medical attention is sought within several hours after ingestion or if the patient has been intubated, removal of the ingested plant by gastric lavage can be considered. Emesis may be induced by using syrup of ipecac if the patient is awake and relatively alert. The usual dose of ipecac is 30 mL for adults and 15 mL for children.[3,5]

After initial assessment and attempts to eliminate the toxin from the gastrointestinal tract, most cases of jimson weed poisoning can be managed simply with observation until symptoms resolve. However, cardiac monitoring, serial recording of vital signs, and serial neurologic assessment are important for detecting occasional occurrence of life-threatening events and for establishing resolution of symptoms. Serial examinations usually indicate improvement within 24 hours, and most patients need less than 48 hours of observation.[10]

Patients with anticholinergic poisoning should be observed by using a cardiac monitor because of the risk for tachyarrhythmia from inhibition of vagal effect on the sinoatrial node.[9] Propanolol may be used for treating symptomatic tachyarrhythmia; the dosage for adults is 1 mg given intravenously for one minute and repeated every five minutes (maximum dose, 5 mg); the dosage for children is 0.01 to 0.1 mg/kg, (maximum dose, 1 mg).[3]

Patients also need close observation for hyperpyrexia and convulsions, because either condition can be fatal.[5] Cooling measures (eg, sponging or a cooling blanket) may be used to treat hyperpyrexia, and intravenous fluid resuscitation may prevent this complication. Convulsions may be treated initially with benzodiazepine therapy.[5] Hypertension is usually transient and usually does not necessitate pharmacologic intervention unless hypertensive crisis is suspected.[9]

In severe cases in which patients have symptoms of anticholinergic crisis (eg, dysrhythmia, coma, seizures, clinically significant hypertension, or poorly controlled hyperpyrexia), the use of physostigmine is

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warranted.[5] Physostigmine is an acetylcholinesterase inhibitor and can therefore reverse the peripheral and central manifestations of anticholinergic excess.[11] The initial dose of physostigmine is 0.5 to 2 mg in adults or 0.02 mg/kg in children, to whom the drug is given slowly by intravenous route. The maximum dose in adults should not exceed 4 mg in 30 minutes.[3] Clinicians must remember that use of physostigmine carries risks and that excess acetylcholine may induce a cholinergic crisis, symptoms of which include bradycardia, complete atrioventricular block, asystole, emesis, bronchorrhea, and seizures.[5] If overcorrection is suspected (eg, as manifested by cholinergic symptoms), 0.5 mg of atropine may be given intravenously for every 1 mg of physostigmine given.[9]

Clinicians must remember that drugs with anticholinergic properties (eg, some antipsychotic and sedative drugs) can worsen symptoms of jimson weed poisoning.

Routine use of physostigmine to treat jimson weed intoxication remains controversial. Closely monitored use of physostigmine in very small doses to prevent cholinergic excess may be safe: When used to treat a series of 23 patients with hallucinations from jimson weed intoxication, physostigmine had no adverse effects.[11] Physostigmine can quickly reverse signs and symptoms of central and peripheral nervous system dysfunction and can assist diagnosis of anticholinergic excess.[12] However, most cases of jimson weed poisoning have a benign outcome after treatment with only supportive care and observation; use of physostigmine is therefore not routine and should be reserved for patients who have clinically significant symptoms or complications.

Benzodiazepine therapy is the main treatment for acute agitation, and use of restraints may be necessary to avoid injury to the patient or hospital staff. Clinicians must remember that drugs with anticholinergic properties (eg, some antipsychotic and sedative drugs) can worsen symptoms of jimson weed poisoning. Agents such as haloperidol or chlorpromazine can exacerbate agitation, and psychosis and should therefore be avoided.[12]

Conclusion

Jimson weed poisoning produces classic anticholinergic symptoms, is usually self-limiting, and usually requires only supportive measures and observation. Recognizing the signs and symptoms of anticholinergic poisoning can help clinicians identify the toxidrome early and intervene appropriately in life-threatening cases, which occur rarely. High levels of jimson weed ingestion may produce dangerous medical conditions, such as cardiac arrhythmia, hyperpyrexia, seizures, coma, and respiratory arrest. Physostigmine is the preferred treatment for severe cases of jimson weed poisoning, and benzodiazepine therapy is the preferred treatment for agitation. Anticipatory counseling, especially around summer and early fall (when the jimson weed plant matures), may help deter adolescents from experimental use of this plant. ❖

Practice Tips
Jimson weed is popular among adolescents curious about the plant's hallucinogenic effects.
Anticipatory counseling for teenagers and parents may also prevent experimentation and resultant harm.
Jimson weed reaches a height of five feet and consists of large, jagged leaves and trumpet-shaped white or purple flowers.
Intoxication results in anticholinergic effects. Initial symptoms include restlessness, shaking, hallucinations, dry mucous membranes, thirst, dilated pupils, blurred vision, and difficulty speaking and swallowing.
The mnemonic for anticholinergic symptoms—"blind as a bat, dry as a bone, red as a beet, mad as a hatter, and hot as a hare."
Treatment may include: activated charcoal, emesis, cardiac monitoring, propanolol, cooling measures, benzodiazepine and physostigmine.

Acknowledgment

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