CASE REPORT

Jimson Weed Intoxication in Five Adolescents

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INTRODUCTION

Datura stramonium (jimson weed) is a poisonous shrub that grows wildly throughout the United States with a high potential for abuse. The plant possesses potent anticholinergic properties, and ingestion can cause serious illness or death. Intentional ingestions may result in unintended poisonings for people who attempt to experience the anticholinergic-induced delirium that typically manifests after ingesting the leaves, stem, seeds, or tea brewed from the leaves. We report 5 cases of *D. stramonium* intoxication seen within a 3-day span as well as recent data regarding anticholinergic plant exposures.

CASE REPORTS

Five males, aged 15-17 years, were admitted to our institution with *D. stramonium* poisoning. Three initial patients (Cases 1-3) presented together to the emergency department 6 hours after ingesting *D. stramonium* seeds. Cases 1-3 presented with the brother of Case 3, who denied personal ingestion of *D. stramonium*. The brother brought them to the emergency department (ED) because he was concerned about the adolescents' incoherent speech, hyperactivity, and incoordination; Case 1 had fallen down some stairs. He explained that friends had instructed them to consume "Datura" or "moonflower" in order to achieve a high, and he presented a bag of seeds that Cases 1-3 had been ingesting. The friends had told them where to find it growing wildly and to consume the contents of "2 pods" each. After admission of Cases 1-3, social workers warned the residents of the group home where Case 3 resided about the dangers of *D. stramonium*. Two days later, however, Cases 4 and 5 (also residents of the group home) presented to the ED 3 hours after ingesting seeds.

All cases presented with disorientation, visual hallucinations, mydriasis, and dry mucous membranes. Basic laboratory values and electrocardiograms were normal for all cases. There were no interval width abnormalities. Cases 1, 4, and 5 required sedation with intravenous lorazepam in the ED. All cases were admitted to the pediatric intensive care unit (PICU) where they were monitored and given intravenous fluids. *D. stramonium* ingestion was diagnosed in all cases by either direct admission from the patient or by first-hand acknowledgment by witnesses; therefore, definitive diagnosis by gas chromatography/mass spectroscopy was not performed. Urine toxicology screens were instituted to rule out additional etiologies for presenting symptoms. Concomitant marijuana use was significant for Cases 1 and 2 only.

Case 1

A 16-year-old male presented with combativeness and unintelligible rambling speech. He had fallen down a flight of stairs while under the influence of *D. stramonium* before presenting to the ED. He attempted to bite and kick staff members and was subsequently sedated. Blood pressure was 143/37, and pulse was 128 beats per minute. Activated charcoal administration was deferred secondary to combativeness. He had a prior history of marijuana use, and urine toxicology screen was accordingly positive. Sinus tachycardia persisted for 14 hours, with a range of 90-128 beats per minute. Discharge from the PICU occurred after 24 hours with normal vital signs and mental status.

Case 2

A 17-year-old male presented with disorientation and visual hallucinations. He was noncombative, and speech was coherent. Blood pressure was 154/70, and pulse was 87 beats per minute. He had muscle incoordination but could follow simple commands. Activated charcoal was administered. Urine toxicology screen was positive for marijuana. At no time was sedation required. Discharge

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from the PICU occurred after less than 24 hours with normal blood pressure and mental status.

Case 3
A 16-year-old male presented with slurred, unintelligible speech and severe restlessness. Blood pressure was 130/67, and pulse was 85 beats per minute. Activated charcoal was administered. Shortly after admission to the PICU, he became aggressive, combative, and even attempted to stand on a bedside table and fly to escape from the room. Subsequently, he was sedated with intravenous lorazepam. Discharge occurred after 24 hours with normal vital signs and mental status.

Case 4
A 15-year-old male was brought to the ED by the police after he had been found crawling down a busy street into an intersection. He was picking at objects on his body that were not present. His only comprehensible words were obscenities. Blood pressure was 119/44, and pulse was 154 beats per minute. Skin was dry and flushed. Urine toxicology screen was negative. Activated charcoal was administered. Blood pressure and temperature remained stable. Pulse ranged from 40 to 109 beats per minute with sinus rhythm. He was sedated as necessary and discharged 40 hours later with normal vital signs and mental status.

Case 5
A 17-year-old male presented with belligerent, unintelligible speech and blurred vision. Blood pressure was 153/77, and pulse was 104 beats per minute. Activated charcoal was administered. Urine toxicology screen was negative. Sedation was continued as necessary. Hypertension and tachycardia resolved without intervention, and he was discharged 24 hours after admission with normal mental status.

DISCUSSION

D. stramonium is a member of the solanaceae family and contains the alkaloids atropine, hyoscyamine, and scopolamine, which can cause severe anticholinergic toxicity when any part of the plant is ingested.[1] Native to the United States, *D. stramonium* is also known as jimson weed, Jamestown weed, thornapple, apple of Peru, angel's trumpet, locoweed, green dragon, trumpet lily, and stinkweed. The plant may grow to 6 ft in height with dark green leaves, a green to purple-green stem, and white or violet funnel-shaped foul-smelling flowers that blossom in the spring. By autumn, ovoid, spiny seed-containing pods develop that are approximately 2 in long and contain brownish-black seeds.[2-5]

Symptoms of classic anticholinergic toxidrome occur within 60 minutes of ingestion and typically include acute hypertension or hypotension, tachycardia, tachypnea, hyperpyrexia, visual hallucinations, and mental status changes. Urinary retention, dysphagia, and dysarthria may also occur. Physical examination may show mydriasis, photophobia, dry mucous membranes, dry and flushed skin, hypoactive bowel sounds secondary to slowed gastrointestinal motility, and muscle incoordination.[1-6] Effects typically last up to 48 hours based on the dose and gastrointestinal impairment.[3] Fatalities are rare, and most patients recover with only supportive care.[1-5]

Anticholinergics competitively inhibit the binding of acetylcholine to the postganglionic parasympathetic muscarinic receptors and the muscarinic receptors in the central nervous system, blocking function of smooth muscle in the eye, gastrointestinal tract, and urinary bladder. Also blocked is the ability to regulate sweat, salivary, and mucosal gland activity.[7] Competitive inhibition of the cardiac cholinergic receptors by the toxin can alter heart rate and conduction through the AV node, often producing sinus tachycardia. Inhibition of central muscarinic receptors impairs cognition, motor coordination, and perception.[7] Ingestion of *D. stramonium*, therefore, results in symptoms that are classic of anticholinergic poisoning: *hot as a hare, blind as a bat, red as a beet, dry as a bone, and mad as a hatter*.[7]

Treatment for anticholinergic toxicity is largely supportive. Gastric lavage should be considered immediately upon presentation,[6] but can be of use as late as 48 hours after ingestion due to slowed gastrointestinal motility.[4] This should be followed by administration of activated charcoal if the patient has a protected airway.[6] Agitation can be controlled by titrating doses of intravenous benzodiazepines to the desired effect. Comatose or severely obtunded patients should be intubated and mechanically ventilated.[8]

Physostigmine, an anticholinesterase inhibitor that acts in both the peripheral and central nervous systems to antagonize muscarinic inhibition, may be used in the management of pure anticholinergic overdose. Its use, however, has been controversial due to adverse outcomes associated with tricyclic antidepressants and electrocardiogram abnormalities.[9,10] The use of physostigmine is indicated when both peripheral (dry mucosa, flushed skin, mydriasis, slowed gastrointestinal motility, urinary retention, and tachycardia) and central (agitation, delirium, hallucinations, seizures, and coma) anticholinergic manifestations are present in the absence of QRS prolongation on electrocardiogram.[5] It should

be given slowly, with an initial dose of 1-2 mg for adults and 0.02 mg/kg (max of 0.5 mg) for children. The dose may be repeated in 10-15 minutes.[5] It should not be given as a continuous infusion.[7] Adverse effects of physostigmine are secondary to acetylcholine accumulation and include seizures, muscle weakness, bradycardia, lacrimation, salivation, bronchorrhea, diarrhea, and asthma exacerbation. It may cause asystole in patients who have taken tricyclic antidepressants.[9] Conduction abnormality demonstrated on EKG is a contraindication to its use.[7] Relative contraindications include asthma, gangrene, coronary artery disease, and mechanical obstruction of the gastrointestinal or genitourinary tracts.[8] Most patients do well, however, with sedation, hydration, charcoal therapy, and observation.[7]

In regard to Cases 1-5, sedation, hydration, charcoal therapy, and observation were sufficient treatment modalities. Sedation requirements ranged from 0 to 8 mg of lorazepam in the ED followed by further incremental dosing as needed. Physostigmine was not administered in any of the cases because the desired therapeutic effect was achieved with sedation. All cases safely returned to normal mental status within a maximum of 40 hours.

An average of 1044 cases of total anticholinergic plant exposures (including *D. stramonium*) have been reported per year in the United States from 1997 through 2001. Fifty-three percent of the cases were treated in health care facilities. Forty-six percent of the ingestions were intentional. Of all cases from 1997 through 2001, 77% occurred in individuals <19 years of age, with 46% of the total cases occurring in patients 6-19 years. Six deaths were reported due to anticholinergic exposures for this time period.[11-15]

CONCLUSION

In this case study we report that *D. stramonium* is readily available to adolescents in our community and that cases of abuse are likely to occur in clusters. As evident by the high percentage of US cases of anticholinergic plant ingestions by people <19 years of age, health care workers must remain aware that anticholinergic plant ingestions (including *D. stramonium*) are common in the adolescent age group. We must consider anticholinergic ingestions in the differential diagnosis for the adolescent that presents with altered mental status.

REFERENCES

1. Rosen CS, Lechner M. Jimson-weed intoxication. *N Engl J Med*. 1962;267:448-450.
2. Mikolich JR, Paulson GW, Cross CJ. Acute anticholinergic syndrome due to Jimson seed ingestion. clinical and laboratory observation in six cases. *Ann Intern Med*. 1975;83(3):321-325.
3. Ellenhorn M. *Ellenhhorn's Medical Toxicology: Diagnosis and Treatment of Human Poisoning*. 2nd ed. Baltimore: Williams and Wilkins;1997:306, 324, 840-845, 1865-1866.
4. Vanderhoff BT, Mosser KH. Jimson weed toxicity: management of anticholinergic plant ingestion. *Am Fam Physician*. 1992:46(2):526-530.
5. Goldfrank, L. *Goldfrank's Toxicologic Emergencies.* 7th ed. New York: McGraw-Hill; 2002:544-547, 1129-1158.
6. Coremans P, Lambrecht G, Schepens P, Vanwelden J, Verhaegen H. Anticholinergic intoxication with commercially available thorn apple tea. *J Toxicol Clin Toxicol.* 1994;32(5):589-592.
7. Delaney, K. Anticholinergics. In Rosen, P. *Emergency Medicine Concepts and Clinical Practice*. 3rd ed. St. Louis: Mosby Year Book;1992:2534-2540.
8. Rumack BH. Anticholinergic poisonings: treatment with physostigmine. *Pediatrics*. 1973;52:449-551.
9. Pentel P, Peterson CD. Asystole complicating physostigmine treatment of tricyclic antidepressant overdose. *Ann Emerg Med.* 1980;9(11):588-590.
10. Suchard JR. Assessing physostigmine's contraindication in cyclic antidepressant ingestions. *J Emerg Med.* 2003;25(2):185-191.
11. Litovitz TL, Klein-Schwartz W, Dyer KS, Shannon M, Lee S, Powers M. 1997 Annual Report of the American Association of Poison Control Centers Toxic Exposure Surveillance System. *Am J Emerg Med*. 1998;16(5):482.
12. Litovitz TL, Klein-Schwartz W, Caravati EM, Youniss J, Crouch B, Lee S. 1998 Annual Report of the American Association of Poison Control Centers Toxic Exposure Surveillance System. *Am J Emerg Med*. 1999;17(5):474.
13. Litovitz TL, Klein-Schwartz W, White S, et al. 1999 Annual Report of the American Association of Poison Control Centers Toxic Exposure Surveillance System. *Am J Emerg Med*. 2000;18(5):559.
14. Litovitz TL, Klein-Schwartz W, White S, et al. 2000 Annual Report of the American Association of Poison Control Centers Toxic Exposure Surveillance System. *Am J Emerg Med.* 2001;19(5):381.
15. Litovitz TL, Klein-Schwartz W, Rodgers GC Jr, et al. 2001 Annual Report of the American Association of Poison Control Centers Toxic Exposure Surveillance System. *Am J Emerg Med.* 2002;20(5):436.