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CASE REPORT

E.P.C.M.S.

Altered Mental Status Fun or Poison!

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ABSTRACT

Datura stramonium is a potentially hallucinogenic plant that grows wild along roadsides, waste areas, desert and tropical regions that has been connected with witchcraft, and traditional medicine. While many names have been given to this plant; the most common name is jimsonweed. All parts of the plant are toxic, although the taste and shape of this plant makes it unattractive to both man and animals but deliberate use by young adults for its hallucinogenic effects have been widely reported for many years. Ingestion of jimsonweed produces the toxidrome of anticholinergic intoxication. Most cases of jimsonweed poisoning have a benign outcome after treatment with only supportive care; use of physostigmine should be used as a therapeutic and diagnostic tool for complicated cases. Recognizing signs and symptoms can help clinicians evaluate patients presenting with altered mental status due to toxicities and potential risks of the recreational use of these plants.

INTRODUCTION

Altered mental status and delirium pose challenging problems to clinicians in variety of clinical settings including emergency medicine, family practice office, and the Intensive Care Unit. Since the differential diagnosis is wide; a systematic approach is necessary to reach the correct diagnosis and administer appropriate treatment. Datura is the genus for a group of hallucinogenic plants that grow wild along roadsides and waste areas in most regions of North America. One of the more common species is (*Datura stramonium*) known as jimsonweed, or "loco" weed which is found throughout the country mainly in the west and southwest. Datura contains three main toxic alkaloids: atropine, scopolamine and hyoscamine. Consumption of any part of the plant can result in severe anticholinergic toxicity.[1] Clinical symptoms are those seen in atropine poisoning, particularly mydriasis, hyperthermia, and altered mental status often with hallucinations. Hospitalization is required for agitation and combative behavior although symptomatic treatment is usually sufficient.

CASE PRESENTATION

An 18 year old Hispanic male was found by the police with altered mental status and bizarre behavior. Per report, the patient, his brother and a friend were found acting in a strange manner and had reportedly ingested an unknown drug. On arrival to the ER, he was found to be restless, delirious, and combative. His past medical information was limited at the time of evaluation. On exam, his initial body temperature was 105.7 °F, blood pressure of 147/95 mmHg, respiratory rate of 48 breath/minute, and heart rate of 102 beats/minute. The patient was somnolent, not responsive to verbal commands but agitated and flexing all four extremities to noxious stimuli. Pupils were dilated and minimally reactive to light. The mouth and the mucus membranes were dry, and he had no intra nasal signs of drugs inhalation. Cardiac examination was normal except for a mild tachycardia. The rest of his physical exam was largely unremarkable with the exception of multiple abrasions and no evidence of intravenous drug use. Laboratory evaluations showed normal results for his CBC, renal panel and liver function tests. His initial Creatine Kinase (CK)however, was 679. Urine drug screen was positive for cannabinoids and a serum drug screen was negative. EKG showed normal PR interval, normal QRS duration, normal QTC and mild tachycardia. A CT scan of the head was negative for bleed and a CXR revealed no acute cardiopulmonary disease. His Initial management included lorazepam for agitation control, volume resuscitation, and passive cooling. The patient was admitted to Intensive Care Unit (ICU) for observation and monitoring. He remained stable, and his mental status improved over the next couple of hours. Later on, the patient admitted that he, his friends, and his brother had intentionally consumed Jimsonweed as a recreational drug while they were wandering around in the desert. After 24 hours of observation in the ICU, the patient's clinical status improved dramatically, however his CK increased rapidly from 679 to 11500 and then 18000 respectively. He was transferred to the ward for further monitoring of his CK which reached a peak of over 20000. IV fluids, in this case normal saline, was administered to dilute his CK which was rising due to muscle breakdown from agitation, and hyperthermia. After the third day of hospitalization his CK dropped to an acceptable level of 2293, and the patient continued to improve throughout his hospital course. He recovered without any apparent sequelae.

DISCUSSION

Based on the patient's description and clinical presentation, the jimsonweed seeds were believed to be Datura stramonium. These plants are known to contain high concentrations of anticholinergic substances; ingestion can result in anticholinergic intoxication. Because of jimsonweed's hallucinogenic properties it is sought out by teenagers and drug users. Parts of the plant can be smoked, chewed, brewed into

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tea, and the seeds can be eaten. The most common effects of this drug are tachycardia, dilated pupils, blurred vision, dry skin, hyperthermia, myoclonic movements, hallucinations, confusion, and difficulty urinating. While true seizures are rare, coma is common and death may occur, often secondary to severe dehydration, hyperthermia, and/or multiple organ failure. The effects of this drug have classically been described by the phrase "Blind as a bat, mad as hatter, red as a beet, hot as hare, dry as a bone, the bowl and bladder lose their tone, and the heart runs alone". [1,2,3] The plant grows in open habitats around cultivated ground, waste places and deserts. These plants have long histories of hallucinogenic use and have been connected with sorcery, witchcraft, native medicine, and magical-religious rites dating back to 1500 BC and Homer's Odyssey.[8] The name "Jimsonweed" is a contraction of "Jamestown weed"; many soldiers and settlers in Jamestown were poisoned after eating its leaves. The effects of jimsonweed on the central nervous system have been exploited medicinally, recreationally and criminally.[8,9]

Recently, Datura has been used as a recreational hallucinogen in the US, resulting in sporadic cases of anticholinergic poisoning and death. During 2005, a total of 975 cases of anticholinergic plant poisonings were reported to Poison Control Centers in the United States; 566 of these cases were treated in health care facilities.[2] During 1998-2004, a total of 188 reported human exposures were identified by Texas Poison Control Centers. Seventy-six percent of the exposures occurred in June-October, 82% of the cases occurred in males, and 72% of cases occurred in those aged 13-19 years.[3] Internationally, the incidence is unknown. However, cases have been reported in Bulgaria, Germany, Italy, Greece, Jordan, Saudi Arabia, Russia, India Tanzania, Australia, Brazil, Asia, West Africa, Mexico, Chile, and Venezuela, attesting to worldwide distribution of *Datura* species.[2,3,4] Numerous cases of anticholinergic poisoning also have resulted from belladonna alkaloid contaminants in foods, including herbal tea, hamburger, honey, and homemade "moon flower" wine. In Europe the dried leaves from the datura plants were used as treatment for Asthma and cough medicine. In China, jimsonweed was prescribed for diseases of the feet and sedative effects, other cultures used the juice to assist in childbirth, and in many occasions the results were fatal.[5,6,7,8] Other accidental ingestions include misuse as an edible wild vegetable and inclusion in homemade toothpaste, as well as a large epidemic in New York and the eastern US that resulted from heroin contaminated with scopolamine.[8,9]

The effective management of Jimsonweed intoxications starts with stabilization of airways, breathing and circulation. Treatment initially should be focused on supportive care, control of agitation with benzodiazepines and hyperthermia with aggressive cooling measures. Gastrointestinal decontamination with activated charcoal should be considered if the patient's mental status is intact and route of administration of the substance was oral, even after many hours has elapsed because of the anticholinergic effects of delayed gastric emptying and slowed perstalsis.[9,10]

Physostigmine should be considered for severe agitation and delirium that does not respond to benzodiazepines. Physostigmine acts as antidote therapy for anticholinergic poisoning. It is a reversible Acetylcholinesterase inhibitor and crosses the blood brain barrier. It acts not only at muscurinic and nicoyinic sites of the autonomic nervous system, but also nicotinic receptors of the neuromuscular junction. Its duration of action is about 2-4 hours. The recommended dose of physostigmine in adults is 0.2-2mg and should be given by slow Intravenous push generally over 3-5minutes. The initial dose may be repeated after 20-30 minutes if necessary. Physostigmine must be used in pure anticholinergic poisoning and should be administered only in controlled setting because of adverse cholinergic manifestations (I.e., vomiting, diarrhea, abdominal cramps, and diaphoresis). Physostigmine also may produce seizures, a complication frequently reported when administered to individuals with tricyclic antidepressant poisoning. In our case we did not use physostigmine because the patient's agitations were controlled by Lorazepam. However, when using physostigmine, clinicians should be prepared to give atropine for cholinergic crisis and caution should be used when giving physostigmine to patients with reactive airway disease, epilepsy and cardiac conduction abnormalities (prolonged PR and QRS intervals) on ECG analysis. [10,11]

Finally, most cases of jimsonweed poisoning have a benign outcome after treatment with only supportive care and observation. Physostigmine can quickly reverse signs and symptoms of central and peripheral nervous system dysfunction and can assist in the diagnosis of anticholinergic excess.[12] Agents such as haloperidol or chlorpromazine can exacerbate hyperthermia through inhibition of sweating, lower the seizure threshold, and should be avoided. Therefore, benzodiazepine therapy is the main treatment for acute agitation. The short-term and careful use of restraints may be necessary to avoid injury to the patient or hospital staff. A patient with agitation and restlessness may develop Rhabdomyolysis due to wasting muscle cells while fighting the restraints. Serum chemistry and electrolyte analysis may provide clues to the intoxicating agents and coingestants. Clinicians should check creatine kinase (CK) level in patients with psychomotor agitation to rule out any association with rhabdomyolysis and must remember that drugs with anticholinergic properties can worsen symptoms of jimsonweed poisoning.[13]

CONCLUSIONS

Ingestion of the Datura species can result in severe toxicity. Each plant varies in the concentrations of alkaloid substances. For this reason, it is very important for individuals to become aware of the toxicities and potential risks associated with the recreational use of these plants. The controversial use of this plant and the side effects makes it an interesting study in pure anticholinergic symptomatology. Primary care physicians should be informed about the abuse of the Datura plant, and the need to educate at risk patients as to the profound dangers of it's abuse. Anticholinergic excess should be considered in the differential diagnosis for adolescent patients presenting with altered mental status. Early consultation with a toxicologist or poison control center is frequently useful for toxic exposures or ingestions to help in the decision-making with regard to decontamination and therapeutic interventions. This is particularly true with the use of physostigmine in cases of atropine - alkaloid poisoning.

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REFERENCES

1. Do It Now Foundation. Jimson Weed: Fast Facts. June 2007, Catalog no. 525. Available at: www.doitnow.org/pages/525.html.

2. Forrester MB. Jimsonweed (Datura stramonium) exposures in Texas, 1998-2004. J Toxicol Environ Health A. Oct 2006; 69(19):1757-62.

3. Lai MW, Klein-Schwartz W, Rodgers GC, Abrams JY, Haber DA, Bronstein AC. 2005 Annual Report of the American Association of Poison Control Centers' national poisoning and exposure database. Clin Toxicol (Phila). 2006; 44(6-7):803-932.

4. Al-Shaikh AM, Sablay ZM. Hallucinogenic plant poisoning in children. Saudi Med J. Jan 2005; 26(1):118-21.

5. Le Strange, R. A History of Herbal Plants. Arco Publishing Company, Inc., New York, 1977. (pp. 99-100)

6. Lewis WH, Elvin-Lewis MPF. Medical Botany: Plants Affecting Man's Health. John Wiley & Sons, Inc., New York, 1977. (pp. 167, 296, 300)

7. Balikova M. Collective poisoning with hallucinogenous herbal tea. Forensic Sci Int. Aug 14 2002; 128(1-2):50-2.

8. Boumba VA, Mitselou A, Vougiouklakis T. Fatal poisoning from ingestion of Datura stramonium seeds.Vet Hum Toxicol. 2004 Apr; 46(2):81-2.

9. Dewitt MS, Swain R, Gibson LB Jr. The dangers of jimson weed and its abuse by teenagers in the Kanawha Valley of West Virginia. W V Med J. 1997 Jul-Aug;93(4):182- Salen P, Shih R, Sierzenski P, Reed J. Effect of physostigmine and gastric lavage in a Datura stramonium-induced anticholinergic poisoning epidemic. Am J Emerg Med. 2003 Jul; 21(4):316-7.

10. Duke JA. Handbook of Medicinal Herbs. CRC Press, Boca Raton, 2001. (pp. 161-162)

11. Richard C. Dart Medical Texicology. Lippincott Williams & Wilkins, 2004. (pp. 52-53)

12. Shenoy RS. Pitfalls in the treatment of jimson weed intoxication [letter]. Am J Psychiatry 1994 Sep;151(9):1396-7.

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