Datura stramonium poisoning in a child

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Hallucinogenic plant poisoning in children is a significant problem for the emergency physician. We describe the case of a boy who had slurred speech, fever, hallucinations, tachycardia, dilated pupils, confusion and disorientation. He had no history of drug use or toxin intake. All signs and symptoms were improved by supportive therapy within 48 hours. It turned out that the patient had ingested seeds of *Datura stramonium* in a neighbor's garden two days previously. The medical history should be taken repeatedly in cases of unknown etiology, and physicians should keep in mind the possibility that unexplained anticholinergic toxidromes could be the result of exposure to toxic plants, in particular those containing atropine and atropine derivates.

Key words: Datura stramonium, poisoning, child.

Datura stramonium (DS) is a common plant found growing throughout the world. It is called by numerous names, including angel's tears, angel's trumpet, Jimson weed, Indian apple and green dragon[1]. Although the toxicity of DS has in the past been associated with accidental ingestion, more recently its consumption has increased because of its usage on the one hand for its hallucinogenic effects and on the other for allaying flu symptoms[2,3]. Mortality from DS intoxication is rare, but adverse effects after its intake are more frequent. DS contains atropine, scopolamine, hyoscine and hyoscamine which are responsible for its toxicity. The toxins in DS are available in all parts of the plant. When DS seeds are ingested, even small amounts may also cause mild cerebral dysfunction, hallucinations and agitation[1]. The toxins function as competitive receptor antagonists in the central and peripheral muscarinic receptors. Therefore, erythema of the skin, dry mouth, hyperthermia, mydriasis, tachycardia, hypotension, hypertension, urinary retention, intestinal dysmotility, muscle weakness, ataxia, hallucinations, agitation, delirium, convulsions, confusion, coma, cardiovascular collapse and other antimuscarinic effects may occur[1-3]. Herein, we report a five-year-old boy who presented with erythema, vomiting, slurred speech and decreased level of consciousness due to DS. This case is interesting in that it emphasizes the importance of a detailed medical history, as well as for the symptoms, which may be confused with other emergency situations because of the plant-derived toxins.

Case Report

A five-year-old boy was admitted to the pediatric emergency department with fever, sudden difficulty in speech and a decreased level of consciousness. Erythema had appeared on his face and body three hours before his arrival at the hospital. Nausea and vomiting soon developed during the night. Afterwards, he began to experience blurred vision and hallucinations involving distorted images of people. Within an hour of admission, he had begun experiencing dizziness and difficulty in speaking and walking. He had no history of drug use or toxin intake, and his past medical history was unremarkable. His weight was 17 kg, and on admission his vital signs were: temperature 38°C, pulse rate 120 beats/min, respiratory rate 22 breaths/min, blood pressure 110/70 mmHg and oxygen saturation 98% on room air. He was confused and agitated, with disorientation evident. His score on the Glasgow Coma Scale was 12/15; his pupils were 5-6 mm bilaterally, with sluggish response to light. His neurologic

exam was notable for slurred speech and an inability to walk. No other focal neurological deficit was observed, and the remainder of the physical examination was unremarkable. Complete blood counts revealed a hemoglobin level of 12.8 g/dl, white cell count of 10,500/mm^3 (51% granulocytes, 45% lymphocytes, 3% monocytes, 1% stab), and a platelet count of 385,000/mm^3. Other laboratory studies showed normal liver and kidney function, C-reactive protein, serum glucose level, serum electrolytes and urine analysis. There were no nucleated or red blood cells on the analysis of cerebrospinal fluid (CSF). CSF pressure was normal. CSF protein was 9.1 mg/dl, and glucose was 63 mg/dl. The urine toxicology screen was negative for benzodiazepines, barbiturates, ethanol, opiates, marijuana, amphetamines and cocaine. Sinus tachycardia was seen in his ECG; his chest X-ray revealed no pathology. Cranial magnetic resonance imaging (MRI) showed no abnormalities. The patient eventually became more able to recognize his mother during his second day in the hospital. By the morning of the second day, he was able to walk with a normal gait and his slurred speech resolved. When the doctors spoke with his parents about his illness, his mother related the fact that he had been playing in the garden two days ago and might have eaten seeds of a plant which grew up in the neighbors' garden. After the patient's father brought in specimens of the plants, we saw that they were *Datura stramonium* (Fig. 1). After the complete resolution of symptoms within 48 hours post-exposure, the patient said that he had eaten seeds and flowers of the plants while in the garden two days ago.

Discussion

Datura species are members of the *Solanaceae* family and are sometimes grown as ornamental plants[1]. They are used for treatment of chronic bronchitis, asthma and flu, and to reduce pain. While in past years exposure to DS generally resulted either from accidental ingestion or from drinking herbal tea for medicinal purposes, nowadays it is ingested primarily for its hallucinogenic effects[2,3]. Toxicity associated with *Datura* species can be seen in many parts of the world, since they can be grown in Europe, Africa, America and Asia. DS is the most commonly encountered *Datura* species[1]. In our patient, anticholinergic effects, such as skin erythema, vision abnormalities, hallucinations, altered mental status, mydriasis, slurred speech and ataxia were observed; these findings were consistent with DS poisoning.

We evaluated our patient from the perspective of psychiatric and organic causes. If a patient with unexplained fever, difficulty in speaking and walking, hallucinations and confusion presents to the pediatric emergency department, physicians must keep in a mind that these symptoms could be the result of hypoglycemia, cerebral hypoxia, hallucinogenic drugs, plant toxicity, drug withdrawal, metabolic disease, postpartum psychosis, electrolyte disturbances, uremia, hepatic encephalopathy, systemic lupus erythematosus, psychiatric disease or central nervous system abnormalities including intracranial masses, lesions or injury, stroke, central nervous system infection or postictal psychosis. Our patient's clinical symptoms were acute onset, his medical history was unremarkable for epilepsy and previous psychiatric disorders, and his laboratory tests, including toxicology screening, were all normal. Lumbar puncture was performed for possible central nervous system infection; however there was no abnormality in the CSF analysis. Furthermore, no organic cause could be detected on the cranial MRI. The determination of anticholinergic toxicity due to DS ingestion can be difficult due to the variety of signs and symptoms. There is no specific test for diagnosis of DS poisoning. The medical history and physical examination of the patient is essential for the diagnosis of DS poisoning[2-4]. Although our patient's parents were questioned at the outset, it was not initially learned that the patient had ingested DS seeds. We realized that the patient had been exposed to DS only when his medical

Fig. 1. *Datura stramonium* plant with flower.

history was received again from his mother. Therefore, a medical history should be taken repeatedly in cases of unknown etiology to uncover additional information.

Management of intoxication related to DS involves primary supportive care[4,5]. Gastric decontamination and activated charcoal are recommended up to 48 hours after ingestion. The patient should be taken to a quiet environment. Benzodiazepines should be used for convulsion and agitation. If cardiac involvement including tachycardia and hypertension is present, beta blockers can be used. When severe central nervous system and cardiac involvement are observed, physostigmine may be necessary[4,5]. Physostigmine is a colinesterase inhibitor that enhances the amount of acetylcholine in the synaptic cleft. Physostigmine is opted for in cases of patients with evidence of severe toxicity, such as seizures, severe hypertension, severe hallucinations and life-threatening arrhythmias due to severe anticholinergic effects; it may also be useful for diagnosis of anticholinergic toxidromes[4]. Death due to DS intoxication rarely occurs. Small doses of DS may bring about local or systemic toxic effects. Supportive care and observation are the fundamentals of the management of DS poisoning, and physostigmine is indicated for severe cases or symptoms persisting more than 48 hours after ingestion[2-5].

In conclusion, if a patient presents with anticholinergic symptoms including altered mental status, hallucinations, seizures, mydriasis, flushed skin, hyperthermia, tachycardia and tachypnea, emergency physicians should suspect DS intoxication. Diagnosis is generally based on the history and clinical findings. In a patient who has been poisoned by DS, the medical history sometimes may not be complete, and unnecessary procedures such as lumbar puncture and cranial imaging may result. Consequently, it is extremely important to obtain a more detailed medical history in order to be able to recognize potential poisoning.

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