CASE REPORT

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Anticholinergic Syndrome Due to the Use of Datura Stramonium Seeds for Constipation: A Story of Prolonged ICU Stay

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ABSTRACT

Datura stramonium is grown widely in some regions of Turkey. Datura stramonium seed is used in the treatment of various diseases such as constipation and acne and causes anticholinergic toxicity when taken in overdose. In the literature, patients presenting with mild symptoms are the majority, and intensive care follow-up is often not required for these patients. In this case report, we present a 50-year-old male patient who received around 200 Datura stramonium seeds for the treatment of chronic constipation. When the patient admitted to the emergency department, he was unconscious, and his pupils were dilated. The patient who had severe vomiting and contractions was intubated. With the diagnosis of anticholinergic syndrome, physostigmine 0.5 mg (one dose) and 1 mg (two times) were administered to the patient with central nervous system findings. The patient who developed sepsis due to aspiration pneumonia during the follow-up stayed in the intensive care unit for 40 days with the support of mechanical ventilator for 37 days. The patient was discharged after 26 days of treatment in the physical therapy service. Accidental ingestion of Datura stramonium seed used for herbal therapy in high doses may cause life-threatening consequences due to its anticholinergic effects or complications related to these effects.

Keywords: Datura stramonium, anticholinergic syndrome, intoxication, aspiration pneumonia, intensive care unit

Introduction

Datura stramonium (DS) is a member of the belladonna alkaloid family The common names of the plant are locoweed, devil's weed, stinkweed, angel's trumpet, thorn apple (1). DS causes anticholinergic syndrome as it contains atropine, scopolamine and hyoscyamine. Common anticholinergic agents are responsible for 15–20% of hospital admissions up to 40% of intensive care unit admission due to poisoning and 16% of poison centre calls (2). According to the 2015 Annual American Association of Poison Control Centers report (3), there were 1091 anticholinergic agent exposures reported to poison control centers that year, ranking 19th on the Substance Categories with the Greatest Rate of Exposure Increase (Top 25) list. The classic symptoms of DS toxicity are tachycardia, hyperthermia, dry skin and mucous membranes, red skin, visual and speech disturbances, decreased intestinal sounds, urinary retention, agitation, disorientation, and hallucinations. Overall, with early identification and adequate supportive care, the prognosis of anticholinergic toxicity is good. Complications include respiratory failure, cardiovascular collapse, rhabdomyolysis, seizures, coma, permanent disability and death (4). Symptoms often start within one-four hours and can last 24–48 hours. DS seeds can be used in the treatment of acne, eczema, and hemorrhoids as well as for their antispasmodic effect (5). Due to its hallucinogenic properties in young people and aphrodisiac effects in older ages, it can cause intoxication as a result of overdose (6, 7). The range of toxicity is highly variable and unpredictable. Fatal atropine poisoning has occurred after as little as 1–2 mg was instilled in the eye of a young child. Intramuscular injection of 32 mg of atropine was fatal in an adult (8). In this case report, a patient who received a very high dose of DS seeds for constipation was discussed and it was aimed to emphasize the importance of knowing the classical findings of anticholinergic agent intoxication in terms of early diagnosis and treatment.

Case

A 50-year-old male patient with no comorbidities took one tablespoonful (approximately 200 pieces) of DS seeds for treatment of chronic constipation. He experienced dizziness, restlessness, vomiting, and losing of consciousness within two hours after ingestion. He was intubated in the emergency room due to unconsciousness. He had tachycardia and contractions at the physical examination. As a result of the blood analysis performed in the emergency room, the patient's glucose was 171 mg/dl, hemoglobin was 8.1 g/dl, and white blood cell (WBC) was 18.42 x 10^9/L, except these results, laboratory results were normal. Acute pathology was not observed in the brain computerized tomography and diffusion-weighted magnetic resonance imaging (DWI), and focal consolidation and diffuse ground-glass opacities were observed in thoracic computerized tomography. Gastrointestinal decontamination with active charcoal was not administered as the time from ingestion to admission was elapsed (indicated in recent ingestion typically <1 hours). Once the patient was stabilized, he was transferred to intensive care unit (ICU).

The patient's Glasgow coma scale was recorded as 3 points, bilateral pupillary light reflex was negative and midriasis was 3+, blood pressure 135/70 mm/Hg, pulse: 131 beats/minute, fever: 36.8°C. On physical examination, heart sounds were tachycardic and had rhonchus on the right basal segment of the lung. Electrocardiogram showed sinus tachycardia. C-reactive protein level was 103 mg/L and procalcitonin level was 41 ng/mL. Ampisilin sulbactam treatment was continued at a dose of 2x1 g with the diagnosis of aspiration pneumonia (he took his first dose at the other center). With the diagnosis of anticholinergic syndrome (9), 0.5 mg (one dose) and 1 mg (two times) of physostigmine was administered (every four hours). Sedation with 5mg/hour midazolam and 50 mcgr/hour fentanyl was ordered for patient's ventilatory asynchrony. Rocuronium was added for the first 24 hour treatment.

The patient had a persistent fever that started on the second day and continued for 11 days. Although the patient was treated with antibiotics and antifungal therapy in accordance with the results of microbiological culture and antibiogram, his fever persisted. Transthoracic/transoesophageal echocardiography and abdominal ultrasonography were performed to investigate the etiology of fever. As a result of echocardiography, it was reported that the EF was 60–65% and there was no vegetation or thrombus in the heart cavities to explain the fever. No pathology was found to explain the etiology of fever on ultrasonography which was repeated twice.

Due to patient's viscous secretions, secretion plugs had formed in the airways. Bronchoscopy was performed to clean these plugs for many times. Even in the bronchoscopy performed just after aspiration, severe inflammation and granulation tissue in the bronchial tissue were remarkable. During the first month of the patient's follow-up, bronchoscopy was performed for eigth times.

Despite daily sedation interruptions the patient's GCS was not change during the areas first two-week. There were diffusion restriction (hypoxic effect) in the bilateral basal ganglia on brain DWI. His GCS was 6 on the 14th day, and was 11 on the 17th day. Tracheostomy was performed on the 15th day The patient was decanulated on the 37th day. The patient, who was followed up for three more days after decanulation, was transferred to the physical therapy service with loss of strength in the left hand and right foot on the 40th day. The patient, who was followed up in the physical therapy service for 26 days, was discharged after a total of 66 days of hospitalization. Written informed consent was obtained from the patient for publication of this case.

Discussion

In DS poisoning, the typical clinic of anticholinergic syndrome occurs. Decreased gastrointestinal motility delays toxin elimination and can cause symptoms to persist for up to 24–48 hours. Few articles in the literature showed that symptoms are prolonged for up to two weeks (10–12).

The symptoms of our patient started two hours after the DS seeds intake. Each of the DS seeds contains about 0.06–0.1 mg of atropine, so consuming a capsule containing about 100 seeds can lead to severe anticholinergic toxicity (5). Our patient received around 200 seeds, and although he vomited, he was brought to the emergency department by his relatives with a severe toxic picture. In the literature, patients with simple anticholinergic symptoms are more common than cases with severe central nervous system findings. Although a case report of death as a result of DS intoxication cannot be reported, complications related to intoxication (severe liver toxicity, cardiac conduction problems and impaired renal function due to rhabdomyolysis) can be observed (13). In our case, kidney and liver functions were normal, there was no rhabdomyolysis, and dysrhythmia. When the literature on DS intoxications in intensive care is reviewed, there are two articles (14, 15). In a case report describing DS intoxication in a 15-year-old male patient (Datura stramonium toxicity mistakenly diagnosed as "bath salt" intoxication: a case report), it was aimed to emphasize that DS intoxications can be confused with synthetic drug intoxications that do not occur in standard drug screenings. In this case report, the patient was admitted to two emergency services, one ICU and one inpatient psychiatric facility, with a pre-diagnosis of bath salt poisoning, but the diagnosis was made in the last center.

Treatment in anticholinergic poisoning includes providing airway patency, breathing and circulation, gastric decompression and active charcoal administration, supportive therapy and antidote administration when necessary. Close monitoring such as cardiac monitoring, monitoring of vital signs and neurological evaluation are important. In patients with severe symptoms of anticholinergic toxicity (dysrhythmia, coma, convulsions, clinically significant hypertension, and uncontrolled hyperthermia) the use of physostigmine (0.5–2 mg in adults, 0.02 mg/kg iv in children) is recommended (16). In a retrospective study (15) conducted with 17 patients who presented with DS poisoning after taking seven to two hundreds seeds, it was found that removal of DS seeds by nasogastric lavage and administration of physostigmine did not cause a significant change in the duration of hospital stay and ICU need. Although it is stated that nasogastric lavage application is effective in removing the seeds even after many hours (57% of the

patients who have undergone lavage), it is not clearly stated how many hours later the lavage is performed. The fact that 13 of 17 patients were admitted to the ICU and physostigmine was used in only three patients may cause bias in the statistical analysis. In our case, gastric decompression and activated charcoal were not applied because four hours passed when he administered to the emergency department. When he came to our ICU, 24 hours had passed after the seeds were taken. He had tachycardia, was unconscious and his pupils were dilated. Therefore, physostigmine was administered to the patient three times with the diagnosis of anticholinergic syndrome. In the patient who had severe acidosis, aspiration pneumonia was also responsible for the deterioration in the patient's clinic in addition to intoxication. The patient remained on a mechanical ventilator for a long time because of lung injury due to aspiration and recurrent infections during follow-up. The patient was decanulated on the 37th day and transferred to the service on the 40th day.

Conclusion

There are many poisoning cases as a result of the traditional treatments, which are widely preferred in our country. Intoxications are common due to the lack of standard doses of the agents used in traditional treatment and it is clear that doctors should be consulted regarding the indication and dosage of traditional treatments. If anticholinergic symptoms accompany in patients presenting with a history of traditional treatment and changes in consciousness, especially emergency healthcare professionals and intensive care professionals should consider DS seed poisoning.

AUTHOR CONTRIBUTIONS:

Concept: SK, KG x; **Design:** SK, ST, MS, KG; **Supervision:** MS, KG; **Materials:**; **Data Collection and/or Processing:** SK, ST, KG, AY, AE, MS, KG; **Analysis and/or Interpretation**: SK, ST, KB, AY, AE, KG; **Literature Search:** SK, ST, KG; **Writing Manuscript**: SK, KB, AY, AE, MS, KG; **Critical Review:** SK, MS.

Informed Consent: Written informed consent was obtained from the patient for publication of this case.

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