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Case Report

Datura stramonium Consumption Causing Severe Anticholinergic Toxicity in an Adolescent Male: A Case Report and Review of the Literature

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Abstract

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Datura is a genus of plants belonging to the Solanaceae family which possess potent, toxic, anticholinergic properties. One member of this genus is the Datura stramonium specie, an ubiquitously-growing hallucinogenic plant which has been known by different names – Jimson's weed, Green Dragon, Angel's Tear – in different parts of the world. Though rare, cases of acute intoxication by *D. stramonium* consumption have been reported, with victims often presenting with fulminant anticholinergic symptoms such as: dry skin, mydriasis and tachycardia, often with fatal consequences. Management of such cases is usually dependent on the severity of symptoms at presentation, ranging from supportive or symptomatic care to the induction of anaesthesia with endotracheal intubation and the use of physostigmine. In line with the CARE criteria, we report a case of acute intoxication following *Datura stramonium* consumption in a 14-year old male. We highlight the manner of case presentation and modalities of patient care in an emergency clinical setting and briefly review the available literature on *D. stramonium* intoxication and its pharmacological properties.

ABBREVIATIONS

DS: *Datura stramonium*; IM: Intramuscular; IV: Intravenous; E/U/Cr: Electrolyte, Urea & Creatinine; CNS: Central Nervous System; LFT: Liver Function Test; AST: Aspartate transaminase; ALP: Alkaline phosphatase; RBG: Random Blood Glucose

INTRODUCTION

Datura is a genus of plants belonging to the Solanaceae family (Angiospermae dicotiledoni). Other plants belonging to this family include: mandrake (Mandrogora officinarum), belladonna (Atropa belladonna), henbane (Hyosyamus niger) and tobacco (Nicotiana tabacum). Some other members of the family are edible fruits such as: tomato (Lycopersicon exculentum), pepper (Capsicum annuum) and potato (Solanum tuberosum) [1]. The Datura genus however is comprised of four species – Datura inoxia, Datura metel, Datura arborea and Datura stramonium – all of which possess potent, toxic, anticholinergic properties [2]. Datura stramonium (DS) is a hallucinogenic plant that has been called numerous names, in various parts of the world such as: Jimson's weed, Angel's Trumpet, Angel's Tear, Thorn Apple,

Devil's Trumpet and Green Dragon [3]. The toxic anticholinergic effect of this plant is a result of three tropane-alkaloids it contains, including: atropine, hyoscyamine and scopolamine. It is important to note that while all parts of this plant are toxic, the range of toxicity is variable in different parts such as: seeds, roots and leaves. Toxicity also varies from season to season and depends on the manner in which the plant is ingested i.e. chewed, drank as an extract or smoked [4]. The highest level of toxins is found in the seeds at a level of approximately 0.1 mg of atropine per seed. Due to the ubiquitous distribution of this plant, and the relative obscurity of its toxidromes, ingestion is often an accidental occurrence, however, most reported cases in literature have been those of teenagers, who ingest this plant mainly for its hallucinogenic effect [4]. We report the case of one such 14 year old who presented to our centre following acute DS intoxication, and we proceed to describe the symptomatology, treatment and policy recommendation for the prevention of similar cases.

CASE PRESENTATION

A 14-year old male high school student was presented to the children emergency department with a history of high pyrexia,

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blurred vision, restlessness and disorientation in a confused state. Documentation from the source of referral suggested ingestion of extract from a local plant called "Gegemu", actually Datura stramonium which had been soaked in water for 7 consecutive days. On arrival at the children's emergency department, he was febrile with temperature of 39.3°C, in a semi-conscious state, disoriented with a Glasgow Coma Scale score of 13 (eye opening response: 4, best verbal response: 4, best motor response: 5). He had markedly dilated pupils (4mm/5mm) which responded slowly to light, and there were no signs of meningeal irritation. Sensory and motor system examinations were essentially normal, but he was tachpnoeic, with a respiratory rate of 34 cycles per minute, and his percutaneous oxygen saturation in room air was 94%. He also had tachycardia, with a heart rate of 120 beats per minute. He was found to be hallucinating, restless and in some form of verbal discourse with unseen individuals.

Serum Electrolyte, Urea and Creatinine (E/U/Cr) done at presentation revealed metabolic acidosis, with serum bicarbonate level of 15mmol/L (reference value: 20-30mmol/L); Full Blood Count done revealed relative neutrophilia, with a neutrophil differential count of 79.5%. Liver Function Test (LFT) revealed deranged findings, with markedly reduced total bilirubin of 1.6 g/dL (reference: 6.0 -8.0g/dL), reduced total protein of 5.0g/ dL (reference: 6.0-8.0g/dL), elevated aspartate transaminase (AST) of 41 IU/L (reference: 0 – 37 IU/L) and elevated alkaline phosphatase (ALP) of 186 IU/L (reference: 40-130 IU/L). Random Blood Glucose (RBG) and urinalysis results were normal, Packed Cell Volume was 42% and malaria rapid diagnostic test was negative. Electrocardiography could not be carried out as it was unavailable at the time of presentation and a full toxicology screening could not be done due to financial limitations.

An assessment of acute intoxication by Datura stramonium was made, with a query of pyogenic meningitis (due to the relative neutropenia). He was placed on a 4-point restraint, due to his violent disposition and given intravenous fluids (0.9% normal saline in 5% dextrose) at maintenance. He was given IM Diazepam at 0.3mg/kg stat, IV Ceftriaxone at 2g. IM Haloperidol was prescribed at 2.5mg, in no more than 2 doses within 24 hours, and a urethral catheter was inserted. Both external (tepid sponging) and internal (via IV Paracetamol, administered at 15mg/kg) were used for temperature control. About 24 hours after presentation, the patient became calm, and regained proper orientation in time, place and person. He remained febrile for the next 24 hours untill he was handed over to the child and adolescent psychiatry team for continued management. He denied prior usage of the D. Stramonium extract, claiming this was the first episode and also denied usage of any other psychoactive substance.

DISCUSSION

Datura stramonium (Figure 1), is a lethally toxic plant known locally as "*Gegemu*", in southwest Nigeria. Several local studies have been carried out to establish the prevalence of substance abuse by adolescents in Nigeria, producing values ranging from as low as 3.8% to as high as 40.1% [5-7]. However, while most of the substances surveyed include traditional drugs such as: tobacco, cannabis, caffeine and cocaine, there is a significant proportion of adolescent psychoactive drug users who rely on local plant extracts, of which *D. stramonium* is an important example [8]. It is

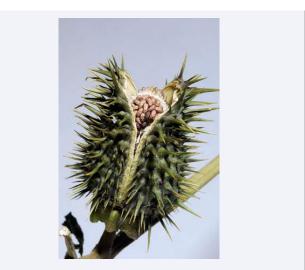
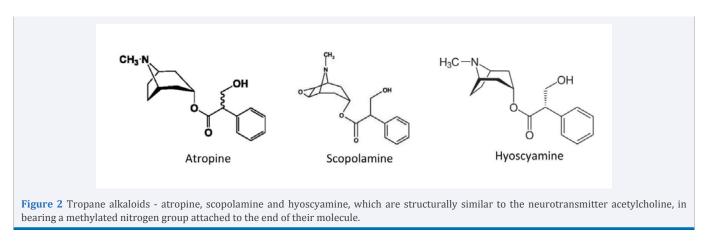


Figure 1 Datura stramonium plant.

also important to note that while ingestion of *D. stramonium* many times occurs accidentally from its local therapeutic use as: antiinflammatory, anti-asthma, anticonvulsant and antihelminthic drug [9], most cases of acute *D. stramonium* intoxication reported in literature are of intentional use by adolescents to experience its hallucinogenic effects. Our presented case was one of such cases of intentional use.

The toxidromes effect of D. stramonium is mediated by its rich content of tropane alkaloids, mainly: atropine, scopolamine and hyoscyamine, which are structurally similar to the neurotransmitter acetylcholine, in bearing a methylated nitrogen group attached to the end of their molecule (Figure 2) [2]. This structural similarity, makes them able to competitively bind to muscarinic cholinergic receptors in the central nervous system, and peripheral synapses, however unlike acetylcholine, they do not stimulate these receptors [10]. This often results in classic anticholinergic symptoms including: dry skin, dry mouth, mydriasis, tachycardia, tachypnea, hypertension, hyperthermia, urinary retention, visual and auditory hallucinations, confusion, disorientation, restlessness and anxiety [11], many of which were described in our patient. In very severe cases, there could be convulsions, coma, respiratory and cardiac failure, eventually leading to death. Rhabdomyolysis, acute kidney injury and fulminant hepatitis have also been reported by Ertekin et al. in 2005 [11]. Our patient also presented with markedly deranged liver function tests, with elevated aspartate transaminase (AST) and alkaline phosphatase (ALP), and reduced bilirubin and total protein. These features appear as early as 1-4 hours after ingestion, and persist for another 24 to 48 hours, due to delayed gastric emptying [12-14]. This is due to the anticholinergic effect of the plant, which causes an inhibition of gastric motility, extending the transit time of the ingested plant in the gastrointestinal tract and consequentially, the duration of the toxidromes. It is also important to note that children are in particular more susceptible to the toxic effects of the plant, and smaller amounts result in more profound CNS and peripheral effects [11]. Reflective of this is the fact that most cases of fatalities from D. stramonium ingestions have been reported in younger children.

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Management of D. stramonium intoxication is mainly supportive and symptomatic. Most patients require hospitalization due to the acutely ill and delirious state in which they present, as well as the risk of organ failure [12,15]. Supportive therapies include: fluid rehydration and oxygen therapy, external (tepid sponging, ventilation) and internal (using antipyretics) temperature control, which were done for our patients. Extreme agitation may be treated using benzodiazepines (IM Diazepam was used for our patient); if presented within 48 hours after ingestion, gastric lavage and activated charcoal may be administered to limit gastric absorption into the blood stream. This was not done for our patient because we could not ascertain the time of ingestion at presentation. In comatose states, rapid sequence induction of anesthesia with endotracheal intubation and mechanical ventilation is recommended [4,10,11]. The specific antidote for D. stramonium intoxication is physostigmine, which acts as a reversible acetylcholinesterase inhibitor, acting at the synapses to increase the concentration of acetylcholine. Because it is also able to cross the blood-brain barrier, it is able to ameliorate the CNS effects of the toxic alkaloids [11]. Physostigmine is administered slowly, at an initial dose of 1-2mg for adults and 0.02mg/kg for children. It may be repeated in 10-15 minutes if sufficient reversal of anticholinergic effects is not achieved [11]. Contraindications for physostigmine use is cardiac conduction defect, particularly the AV block [4]. Physostigmine is often only used in cases of intractable seizures, coma, tachycardia or dysrhythmia and hemodynamic instability, severe agitation and respiratory distress [4]. A retrospective study by Salen et al. [16], in 2003, revealed that physostigmine administration and gastric lavage did not reduce the incidence of intensive care unit admissions or shorten the length of hospitalization for D. stramonium intoxication. Physostigmine was not administered to our patient because it was unavailable in our centre at the time of presentation.

In most cases reported in literature, outcome with proper supportive therapy and physostigmine administration when indicated is generally good. Fatalities have been rarely reported and are due to respiratory arrest or cardiovascular collapse, especially in children [13,14].

In conclusion, a patient who presents with acute anticholinergic symptoms, especially an adolescent, lacking an obvious cause, could suggest *D. stramonium* intoxication and early detoxification should be initiated by the attending physician. For

a plant with such potent toxidromes and ubiquitous distribution, the lack of effective legislation to limit or prevent intentional consumption is a significant legislative loophole. It is important that the public be educated, especially in locations where the plant may be found growing, about its potentially lethal effects.

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