

CASE REPORT

A TRADITIONAL MALAY MYTH LEADING TO UNINTENTIONAL SELF INTOXICATION WITH *KECUBUNG* FRUIT

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Abstract. Traditional Malay herbal medicine is still used in Malaysia especially in rural areas, instead of using modern medicine. *Datura* or "*kecubung*" has been used to treat allergic rhinitis in certain places. Inaccurate doses can potentially cause severe or fatal neurologic anti-cholinergic toxidromes. A good knowledge of toxidromes with optimization of supportive care can prevent fatal complications and lead to a more speedy recovery. We present a case of *kecubung* poisoning.

INTRODUCTION

Malaysia is rich in herbs and plants. In Kelantan state, which is situated in north-eastern peninsular Malaysia, there are 146 identified species of plants used by villagers to treat various ailments (Ong and Nordiana, 1999). They are picked from bushes or jungles as vegetables or "*ulam*" in the Malay language, and sometimes have been used as traditional herbs or medicines to treat certain diseases. In Kelantan, which is predominantly populated by Malays, *datura* is eaten as a traditional medicine to treat allergic rhinitis. We report a serious complication of "*Buah Kecubung*" (scientific name "*Datura*") for the treatment of allergic rhinitis. Interestingly, of the four family members who ate *Datura*, only one person

developed severe anticholinergic symptoms. We present a case report of unintentional *datura* poisoning presenting to the Emergency Department, Hospital of University Sains Malaysia.

CASE REPORT

A 48 year-old gentleman was brought by his two sons in the early evening to the Emergency Department, Hospital Universiti Sains Malaysia with a complaint of dizziness, blurring of vision and altered sensorium. Prior to this, he had been well and ambulating at home. He was able to perform his daily activities. On further questioning, there was no past history of fever, headache, head injury, altered sensorium or epilepsy. There was no history of medical or psychiatric illnesses.

On arrival, he did not respond to calling, he was restless. On the Glasgow Coma Scale he had a verbal score of 3, a motor score of 4 and an eye score of 3. His pupils were 4 mm bilaterally and responded sluggishly

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to light. His neck was stiff with increased reflexes and had equivocal plantar reflexes. He was tachypneic with a distended bladder. His pulse rate was 100 bpm, blood pressure was 160/94 mmHg, axillary temperature was 38°C, SpO₂ was 98% with oxygen at 3 liters/minute. His face was flushed and he had a dry oral mucosa.

The blood investigation for full blood count, electrolytes and sugar were normal except for leukocytosis. An arterial blood gas revealed normal acid-base balance. His electrocardiography showed sinus tachycardia and his chest x-ray was normal. The initial impression was altered sensorium with a reduced general conscious score (GCS) of 10/15 secondary to meningitis, to rule out a space occupying lesion. High flow oxygen (15 liters/minute) and fluid resuscitation were initiated. He was sedated with midazolam at 0.5 mg/hour. Computed tomography (CT) of the brain with contrast was performed but was normal finding.

After 2 hours struggling with a diagnosis, further detailed history from his daughter revealed he ate three datura "*ulam*" with his late lunch, 2 hours prior to presentation. One hour later, he had muscle weakness associated with altered sensorium, then fell to the floor. He was treated as having acute anti-cholinergic toxicity due to datura poisoning. Nasogastric lavage was performed and a slow bolus of neostigmine 2 mg was administered. The first dose of activated charcoal [0.5 g/kg (30 g)] was given in the emergency department before transferring the patient to the high dependency ward. Subsequently, he developed sinus bradycardia of 40 bpm which required atropine 0.5 mg resuscitation. His vital signs were stable until 4 hours later when he developed sinus tachycardia. A second dose of neostigmine 2.5 mg was given. Sixteen hours after admission he regained full consciousness and was discharged home on Day 3 without compli-

cations. The other 3 family members who shared one datura *ulam* were well without any specific complaints. They denied symptoms of anti-cholinergic toxicity.

DISCUSSION

The plant genus *Datura* belongs to the family of Solanaceae or "*Kecubung*" in Malaysia or "*Trompetbloem*" in Surinam. Its leaves are dried and smoked like cigarettes in the treatment of respiratory conditions. A paste of pounded leaves has been applied to the skin to treat aching bones. The smoke from burning the fruit is directed into the mouth to relieve toothache. *Datura* is rich in alkaloids and terpenes and is mostly used for pulmonary conditions such as bronchial asthma (Ong and Nordiana, 1999; Abdolali *et al*, 2006).

Datura is a woody-stalked, leafy herb growing up to 2 meters. It produces spiny seed pods and large white or purple trumpet-shaped flowers that face upward. It has a long history of use both in South America and Europe and is known for causing delirious states and poisonings in users. Its species are *ceratocaula*, *inoxia* (meteloides), *stramonium* and *brugmansia* (Tree Datura). In this case the patient ingested the fruit of *Datura stramonium*, a green-stemmed, hairless annual, 2 to 4 feet tall, with a few branches and two 8 inch long ovate leaves. The flowers are white and 4 inches long. The capsule (fruit) is egg-shaped, up to 2 inches long and filled with many black seeds (www.erowid.org/plants/datura/datura.shtml). The seeds are the most toxic part of the plant as 100 seeds contain the equivalent of approximately 6 mg of atropine (Shervette *et al*, 1979). The main active chemicals present in *Datura* are the tropane alkaloids scopolamine, atropine, and hyoscamine (Bruneton, 1999). Scopolamine and atropine are anticholinergic deleriants. They block muscarinic

receptors, which in turn excite dopaminergic neurons. They are readily absorbed, partially metabolized by the liver, and mostly eliminated in urine, with a half-life of about four hours.

Instead of using *datura* as an abused hallucinogen as reported in western countries, it has been used by traditional healers to treat allergic rhinitis in Kelantan. Its anticholinergic and antimuscarinic effect, atropine and other anticholinergic substances competitively block the action of acetylcholine at muscarinic receptors both centrally and peripherally at end organ sites of the parasympathetic nervous system (Philip *et al*, 2003). Muscarinic receptors affect smooth muscle function of the eye, intestinal tract, urinary bladder, and sweat, salivary and mucosal gland activity. Cardiac cholinergic receptors are present in vagal nerve fibers and affect conduction through the atrioventricular node. Muscarinic receptors in the central nervous system play a role in information storage, cognition, and motor coordination. Toxicity is characterized by high body temperature, delirium, hallucinations, agitation, and persistent memory disturbances. At low doses, it can cause mucosal dryness, which can relieve nasal congestion which improves allergic rhinitis symptoms. At other doses the toxic symptoms ranges from mild to severe. Severe intoxication may cause flaccid paralysis, convulsions, and death (Philip *et al*, 2003). The dose related symptoms explain why the other 3 family members did not manifest with any symptoms.

Our patient manifested with a severe form of intoxication. His blood atropine level was not measured because our laboratory cannot determine the atropine level. Our diagnosis was based on history and clinical presentation, recognising the anticholinergic symptoms. Our approach to management was by giving supportive care

and gastrointestinal decontamination with activated charcoal. In this case, 4 hours after ingestion, gastric lavage is a controversial issue. Considering its active metabolites cause slowing of gastrointestinal peristalsis and the physical characteristics of the seeds which are small in size and difficult to digest, we decided to perform nasogastric lavage. Gastric lavage may play an important role in removing the remaining particles and active solution in the gut, especially since the substance is absorbed slowly. Gastric lavage usually should not be considered unless a patient has ingested a potentially life-threatening amount of a poison and the procedure can be undertaken within 60 minutes of ingestion (Edward, 1998).

Multiple doses of activated charcoals (MDAC) are another method for gastric decontamination and have been advocated to increase the systemic clearance of many drugs. Its role is to prevent further absorption of the toxin into the systemic circulation by adsorbing the toxin by the active charcoal (Jeffrey *et al*, 1987). Routinely administered to reduce gastrointestinal (GI) absorption of many drugs, growing evidence indicates that repeated doses of charcoal may also enhance drug elimination. MDAC is an indicated therapy and is more effective for drugs with a longer intrinsic half-life (Jeffery and Peter, 1992). While supportive management remains the mainstay of therapy in poisoned patients, activated charcoal is inexpensive, effective, simple to administer and may obviate the need for more invasive methods of toxin removal.

The role of neostigmine as an acetylcholinesterase inhibitor in anti-cholinergic intoxication is reported to reverse the effect of anti-cholinergic toxicity (Burns *et al*, 2000). The doses and frequency of neostigmine administration can be titrated according to the clinical symptoms (Beaver and Gavin, 1998). Our patient seemed to respond well

to the regimen given. Too high doses of neostigmine can cause severe bradycardia, which requires administration of intravenous atropine 0.5 mg to improve the heart rate.

The improvement in this patient's central nervous system symptoms by regaining consciousness by 16 hours is fast compared to previous observations. This may be due to the supportive and preventive measures performed in this patient. When taken orally, the effects of *Datura* can begin in 20 to 30 minutes or as long as 2 to 3 hours, depending on the dose ingested. When smoked, the effects may begin as quickly as 5 minutes. A moderate *Datura* dose may last for 8 to 12 hours, while strong doses can cause effects lasting for 2 to 3 days (www.erowid.org/plants/datura/datura_basics.shtml).

In conclusion, early detection of toxicity and a good history remain the most important elements in the diagnosis of datura toxicity in the emergency department (Nasir *et al*, 2009). Delay in recognizing the toxicity of datura may lead to serious morbidity or even mortality.

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