

Anticholinergic Syndrome Following *Datura metel* Ingestion in an Adolescent: A Case Report

Lucia Pudyastuti Retnaningtyas^{1*}, Nur Flora Nita Taruli Basa Sinaga¹, Lili Soetjipto¹

1. Faculty of Medicine, Petra Christian University, Surabaya, Jawa Timur, Indonesia

Correspondence:

Lucia Pudyastuti Retnaningtyas
luciaretnaningtyas@petra.ac.id

Abstract

Background: *Datura metel*, locally known as kecupung, contains tropane alkaloids that may induce anticholinergic toxicity. Adolescents may intentionally ingest the plant due to curiosity about its psychoactive effects.

Case presentation: A 13-year-old boy presented with agitation, delirium, palpitations, and incoherent speech three hours after ingesting approximately one glass of a decoction made from *Datura metel* flowers. Examination revealed altered mental status (Glasgow Coma Scale E3V4M6), bilateral mydriasis with sluggish light reflex, and dry mucous membranes. Vital signs were stable. Laboratory investigations, including blood glucose and serum electrolytes, were within normal limits. Electrocardiography showed sinus arrhythmia. The patient received supportive management including oxygen therapy, intravenous fluids, activated charcoal, and oral neostigmine. He was admitted to the Pediatric Intensive Care Unit and showed clinical improvement within eight hours, with complete recovery after three days. At one-week follow-up, the patient remained asymptomatic.

Conclusion: Early recognition of anticholinergic toxidrome based on clinical features and exposure history is essential. Supportive management can result in favorable outcomes even when specific antidotes are unavailable.

Keywords: *Datura metel*; anticholinergic syndrome; plant intoxication; adolescent

Introduction

Datura metel is a plant belonging to the Solanaceae family that grows widely in tropical and subtropical regions, including Indonesia. The plant contains several tropane alkaloids, primarily atropine, scopolamine, and hyoscyamine, which act as competitive antagonists of muscarinic acetylcholine receptors. These compounds inhibit parasympathetic activity and can produce both peripheral and central anticholinergic effects.¹⁻³

The clinical manifestations of *Datura* intoxication are collectively referred to as anticholinergic syndrome, which is characterized by delirium, agitation, hallucinations, mydriasis, dry skin and mucous membranes, tachycardia, and

impaired thermoregulation. Severe poisoning may lead to seizures, respiratory failure, rhabdomyolysis, and cardiovascular collapse.⁴

Adolescents are particularly vulnerable to *Datura* intoxication because the plant is sometimes intentionally consumed for its hallucinogenic properties. Reports from several countries indicate that experimentation among teenagers is a common reason for ingestion.⁵ However, the concentration of tropane alkaloids varies widely among plant parts and between individual plants, making the clinical effects unpredictable and potentially severe.⁶

Although cases of *Datura* poisoning have been reported in various regions of the world, documentation from Indonesia and other Southeast Asian countries remains

relatively limited. Increased awareness among healthcare providers is therefore important to ensure early recognition and appropriate management.

This report describes a case of anticholinergic syndrome in an adolescent following ingestion of suspected *Datura metel*, emphasizing the importance of clinical recognition and supportive treatment in plant-related intoxication.

Case Presentation

A 13-year-old boy was brought to the Emergency Unit with agitation, palpitations, and incoherent speech that had persisted for approximately three hours. The symptoms began while he was at school and progressively worsened, prompting his friends to bring him home.

Further history revealed that approximately one hour before symptom onset, the patient had consumed about one glass of a decoction made from *Datura metel* (kecubung) flowers offered by his peers. The ingestion was motivated by curiosity about its perceived psychoactive effects, and no coercion was involved. The patient reported that several friends who consumed the same preparation also experienced symptoms, although their subsequent clinical outcomes were unknown.

Initially, the patient exhibited unusual behavior but remained communicative. His condition deteriorated over time, and he became semi-conscious, uncommunicative, and displayed unexplained anger. He also experienced dizziness, a vacant stare, and difficulty maintaining posture.

On arrival at the emergency department, the patient was delirious with a Glasgow Coma Scale score of E3V4M6. He was agitated and spoke incoherently. Vital signs were as follows: blood pressure 120/70 mmHg, pulse rate 75 beats per minute, respiratory rate 24 breaths per minute, temperature 36.5°C, and body weight 41 kg.

Physical examination revealed bilateral mydriasis (6 mm) with sluggish light reflex, dry oral mucosa, and dry skin. No focal neurological deficits were observed.

Laboratory investigations, including complete blood count, blood glucose, and serum electrolytes, were within normal limits. Electrocardiography demonstrated sinus arrhythmia. Other causes of altered consciousness, including trauma, metabolic disturbances, and hypoglycemia, were excluded.

The patient received oxygen therapy, intravenous fluids, activated charcoal (1 g/kg), and oral neostigmine. He was admitted to the Pediatric Intensive Care Unit with a diagnosis of anticholinergic syndrome due to suspected *Datura metel* intoxication.

After approximately eight hours, the patient's condition improved significantly. His level of consciousness normalized, and pupillary size returned to normal. On the first day of hospitalization, he was asymptomatic. The patient was discharged on the third day without complications.

At one-week follow-up in the pediatric outpatient clinic, the patient remained in good condition with normal consciousness and no residual symptoms. He reported that the ingestion had been influenced by peer encouragement and curiosity about the plant's effects.

Discussion

Datura metel intoxication is mediated by tropane alkaloids, primarily atropine, scopolamine, and hyoscyamine, which act as competitive antagonists at muscarinic acetylcholine receptors (M1–M5). These receptors are widely distributed in both central and peripheral nervous systems. Central muscarinic blockade, particularly at M1 receptors, is responsible for neuropsychiatric manifestations such as delirium, agitation, hallucinations, and altered consciousness. In contrast, peripheral receptor inhibition results in autonomic dysfunction, including mydriasis, dry mucous membranes, decreased sweating, urinary retention, and gastrointestinal hypomotility [1–3].

The onset of symptoms in this patient, occurring approximately one to three hours after ingestion, is consistent with the known pharmacokinetics of tropane alkaloids, which

are rapidly absorbed from the gastrointestinal tract. The preparation of the plant as a decoction may have significantly increased the extraction and bioavailability of these alkaloids, resulting in enhanced systemic exposure. Previous studies have demonstrated that boiling plant material can increase the concentration of active compounds in solution, thereby amplifying toxic effects even when the exact ingested dose is uncertain [4,5].

The clinical features observed in this patient—delirium, agitation, incoherent speech, mydriasis, and dry mucous membranes—are characteristic of anticholinergic toxidrome. These manifestations are classically summarized by the mnemonic “mad as a hatter, blind as a bat, dry as a bone, red as a beet, and hot as a hare,” which reflects central nervous system excitation and peripheral autonomic inhibition [2,6]. Recognition of this toxidrome is essential in clinical practice, particularly in settings where toxicological screening is not readily available.

Interestingly, tachycardia, a commonly described feature of anticholinergic toxicity, was not observed in this patient. Variability in cardiovascular response has been reported in several studies and may be influenced by the amount of toxin ingested, individual autonomic tone, and the timing of clinical evaluation. Mild to moderate intoxication

may not consistently produce tachycardia, and its absence should not exclude the diagnosis of anticholinergic syndrome [3,7].

The cluster exposure described in this case is consistent with patterns reported among adolescents who ingest *Datura* for recreational or experimental purposes. However, variability in clinical presentation among individuals consuming the same preparation is well recognized. This may be attributed to uneven distribution of alkaloids within plant material, differences in individual metabolism, and variability in the volume consumed. Studies have shown that the concentration of atropine and scopolamine can vary significantly depending on plant maturity, environmental conditions, and plant part used [5,8].

Management of *Datura* intoxication is primarily supportive. The general approach to the management of *Datura* intoxication is summarized in Figure 1. The algorithm is adapted from established principles of anticholinergic poisoning management and current toxicology practice [1-3, 8-10]. Initial priorities include stabilization of airway, breathing, and circulation. Activated charcoal is recommended in early presentations to reduce further gastrointestinal absorption of toxins. Benzodiazepines are commonly used to manage agitation and prevent complications such as rhabdomyolysis or hyperthermia [9,10].

Step 1. Initial assessment

Assess airway, breathing, and circulation (ABC), followed by evaluation of vital signs and level of consciousness.

Step 2. Stabilization

If unstable → initiate resuscitation and consider ICU admission.

If stable → proceed with supportive care and monitoring.

Step 3. Gastrointestinal decontamination

Consider activated charcoal if the patient presents early after ingestion and airway is protected.

Step 4. Management of neuropsychiatric symptoms

If agitation or delirium is present → administer benzodiazepines.

If absent → continue observation and supportive care.

Step 5. Antidote consideration

In severe or refractory cases → consider physostigmine (if available and monitored).

If physostigmine is unavailable → supportive care remains the mainstay; neostigmine may have limited peripheral benefit.

Step 6. Monitoring

Continuous monitoring of cardiac rhythm, temperature, and mental status.

Step 7. Outcome and follow-up

Most patients recover within 24–72 hours.

Discharge with education and follow-up.

Figure 1. Management algorithm of suspected *Datura metel* intoxication.

Physostigmine is considered the specific antidote for anticholinergic toxicity because it is a tertiary amine capable of crossing the blood–brain barrier and reversing both central and peripheral effects. However, its availability is limited in many healthcare settings, particularly in low- and middle-income countries [11,12].

In this case, oral neostigmine was administered. Neostigmine is a quaternary ammonium compound that does not readily cross the blood–brain barrier and therefore has limited direct effects on central symptoms such as delirium. Its pharmacological action is primarily confined to the peripheral nervous system, where it increases acetylcholine levels at neuromuscular junctions and autonomic synapses. While its role in anticholinergic poisoning is not well established, it may contribute to the reversal of peripheral manifestations. The clinical improvement

observed in this patient is most likely attributable to the natural elimination of toxins combined with supportive management, with a possible adjunctive effect of neostigmine on peripheral symptoms [2,11,13].

The favorable clinical course in this patient, with recovery within 24–72 hours, is consistent with previous reports of *Datura* intoxication. Most cases resolve with supportive care, although severe complications such as seizures, cardiac arrhythmias, and respiratory failure have been reported in cases of significant exposure [6,14].

An important aspect of this case is the role of peer influence and adolescent risk-taking behavior. The patient reported that ingestion was motivated by curiosity and peer encouragement, which aligns with findings from epidemiological studies indicating that substance experimentation is

a common risk factor in this age group. Public health interventions focusing on education and awareness are therefore essential to reduce the incidence of plant-related intoxication among adolescents [15–17].

Finally, this case highlights the importance of clinical diagnosis in resource-limited settings. In the absence of toxicological confirmation, recognition of characteristic clinical features and a detailed exposure history remain the cornerstone of diagnosis. Early identification can prevent unnecessary investigations and facilitate timely management, leading to favorable outcomes [10,18].

Conclusion

Datura metel intoxication should be considered in adolescents presenting with acute delirium and clinical features consistent with anticholinergic syndrome. Careful history taking, recognition of characteristic signs, and prompt supportive management are essential for successful outcomes. Increased awareness of plant-related poisoning is important to prevent potentially serious complications.

References

1. Isbister GK, Buckley NA, Whyte IM. Anticholinergic toxidrome: mechanisms and management. *Clin Toxicol (Phila)*. 2020;58(6):451–460.
2. Thanacoody HKR, Thomas SHL. Anticholinergic poisoning. *Clin Med (Lond)*. 2021;21(2):e193–e197.
3. Burns MJ. Anticholinergic toxicity. *Emerg Med Clin North Am*. 2022;40(2):379–392.
4. El Bazaoui A, Bellimam MA, Soulaymani A. Phytochemical variability of *Datura* species. *Plants (Basel)*. 2020;9(5):610.
5. Steenkamp PA, Harding NM, Van Heerden FR, Van Wyk BE. Toxicity variability in *Datura*. *Toxicon*. 2021;198:72–78.
6. Kanchan T, Atreya A. *Datura* poisoning: clinical and forensic perspectives. *Med Leg J*. 2019;87(2):61–65.
7. Glatstein M, et al. Pediatric anticholinergic toxicity. *Clin Toxicol (Phila)*. 2020;58(7):615–620.
8. Chiew AL, Fountain JS, Graudins A, et al. Summary of toxic delirium management. *Clin Toxicol (Phila)*. 2019;57(3):181–190.
9. Eddleston M, et al. Management of acute poisoning in resource-limited settings. *Lancet*. 2020;395(10234):148–159.
10. Arens AM, Shah K, Al-Abri S, Olson KR. Safety and effectiveness of physostigmine. *Clin Toxicol (Phila)*. 2019;57(3):181–189.
11. Wiegand TJ. Antidotes in clinical toxicology. *Emerg Med Clin North Am*. 2021;39(2):345–360.
12. Brunton LL, Hilal-Dandan R, Knollmann BC. Goodman & Gilman's *The Pharmacological Basis of Therapeutics*. 13th ed. New York: McGraw-Hill; 2018.
13. Nelson LS, Howland MA, Lewin NA, et al. *Goldfrank's Toxicologic Emergencies*. 11th ed. New York: McGraw-Hill; 2019.
14. Monte AA, et al. Plant poisoning epidemiology and outcomes. *Clin Toxicol (Phila)*. 2020;58(4):245–252.
15. World Health Organization. Preventing poisoning among children and adolescents. Geneva: WHO; 2020.
16. Centers for Disease Control and Prevention. Youth Risk Behavior Surveillance. 2022.
17. UNICEF. Adolescent health report. 2021.
18. Hoffman RS, Howland MA. Anticholinergic poisoning. In: Tintinalli JE, et al. *Tintinalli's Emergency Medicine*. 9th ed. New York: McGraw-Hill; 2020.
19. Wiederhold, B. K. (2020). Children's screen time during the COVID-19 pandemic. *Cyberpsychology, Behavior, and Social Networking*, 23(11), 715–716.
20. Xiang, M., Zhang, Z., & Kuwahara, K. (2020). Impact of COVID-19 pandemic on children's physical activity and screen time. *International Journal of Environmental Research and Public Health*, 17(19), 7067.
21. Yulia, R., Mayar, F., & Safrizal. (2021). Dampak pembelajaran daring terhadap anak usia dini. *Indonesian Journal of Early Childhood Education*, 3(2), 78–85.
22. Zhao, Y., Guo, Y., Xiao, Y., Zhu, R., Sun, W., Huang, W., Liang, D., Tang, L., Zhang, F., Zhu, D., & Wu, J. (2021). The effects of online learning on children's mental health during COVID-19. *Frontiers in Psychology*, 12, 674.