
SHORT COMMUNICATIONS & CASE REPORTS

Acute anticholinergic syndrome from *Atropa belladonna* mistaken for blueberries

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PURPOSE. *To report the first case in the ophthalmic literature of acute anticholinergic syndrome after ingestion of *Atropa belladonna* mistaken for blueberries.*

METHODS. *A 36-year-old woman presented to our ophthalmic emergency department with complaints of blurry vision, lightning flashes, disorientation, loss of balance, agitation, and anxiety for 24 hours. Ophthalmic examination revealed bilateral pupillary dilatation and paresis of accommodation. Additional symptoms of the anticholinergic syndrome were elicited on further questioning.*

RESULTS. *Anticholinergic intoxication was suspected and the patient admitted to have eaten six "blueberries" found in the forest the previous day. The patient identified *Atropa belladonna* as the source of the berries she had eaten when shown photographs of the plant and its fruit. The recommendations of the Swiss Toxicological Information Centre were followed and physostigmine, the antidote for severe poisoning when 10 or more berries are ingested, was not administered.*

CONCLUSIONS. *Accidental ingestion of *Atropa belladonna* berries may cause patients to first consult an ophthalmologist. It is important to recognize the anticholinergic syndrome caused by such intoxication in order to make a proper diagnosis, avoid unnecessary testing, and provide expedient appropriate treatment when required. (Eur J Ophthalmol 2009; 19: 170-2)*

KEY WORDS. *Accommodative paresis, Anticholinergic syndrome, *Atropa belladonna*, Mydriasis*

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INTRODUCTION

Atropa belladonna, also known as deadly nightshade, is a perennial herbaceous plant with leaves and berries that are highly toxic. It is native to central and southern Europe, North Africa, and southwest Asia, and has become naturalized in England, France, and North America (1).

All parts of the plant contain hyoscyamine, atropine, and scopolamine, which are potent tropane alkaloids (Fig. 1). However, the berries are the greatest danger and have a sweet taste. *Atropa belladonna* ingestion may produce only transient symptoms (1, 2) but death may occur from severe intoxication (3). We report here a case of acute anticholinergic syndrome after ingestion of *Atropa belladonna* mistaken for wild blueberries.

METHODS

A previously healthy 36-year-old woman presented to our emergency department with complaints of blurry vision, lightning flashes, disorientation, loss of balance, agitation, and anxiety for 24 hours. Ophthalmic examination revealed bilateral pupillary dilatation and mild paresis of accommodation. Best-corrected visual acuity was 20/20 for distance and 20/25 for near. Ocular motility, confrontation fields, slit lamp, and fundus examination were normal.

Pharmacologic intoxication was hypothesized and on close questioning the patient admitted to having eaten six "wild blueberries" found in the forest the previous day. The patient identified the *Atropa belladonna* plant as the source of the berries she had eaten when shown pho-

Fig. 1 - (A) *Atropa belladonna* plant. **(B)** *Atropa belladonna* fruit. (Source: Geneva Botanical Garden).



tographs of the plant and its fruit. The patient also admitted to having had tachycardia, flushing, nausea, dry mouth and throat and skin, hallucinations the night before, and difficulty breathing and urinating, which are all compatible with *Atropa belladonna* intoxication.

RESULTS

The recommendations of the Swiss Toxicological Information Centre were followed and we did not prescribe physostigmine, the antidote administered for severe poisoning when 10 or more berries are ingested. We informed the patient that resolution of the nonocular symptoms usually takes 24–48 hours and that ocular symptoms could persist for several days. All systemic and ophthalmic symptoms disappeared 48 hours later.

DISCUSSION

While poisoning from *Atropa belladonna* has been well described, we were not able to find a single case of acute anticholinergic syndrome by *Atropa belladonna* ingestion reported in the ophthalmic literature. This case is also interesting in that our patient first sought care from an ophthalmic emergency service rather than a general medical emergency department.

Ophthalmic, neurologic, and systemic symptoms of *Atropa belladonna* intoxication are those of the acute anticholinergic syndrome, antagonization of acetylcholine at muscarinic receptors. Ophthalmic symptoms include blur-

ry vision, difficulty reading, and dilated pupils. Systemic symptoms include tachycardia, loss of balance, feeling of flight, staggering, sense of suffocation, flushing, husky voice, meaningless speech, auditory and visual hallucinations, vomiting, decreased intestinal motility, dry throat, mouth, and skin, urinary retention, confusion, agitation, and fever. Severe intoxication may lead to lethargy, respiratory distress, convulsions, coma, and death (1, 2). The central and peripheral signs and symptoms from *Atropa belladonna* intoxication are summarized in Table I. *Atropa belladonna* poisoning may be misdiagnosed as delirium tremens or acute psychosis.

Physostigmine is the mainstay of treatment (1, 4). This alkaloid acts as a reversible anticholinesterase, potentiat-

TABLE I - SIGNS AND SYMPTOMS OF ATROPA BELLADONNA INTOXICATION

Central	Peripheral
Altered mental status	Mydriasis
Delirium	Blurry vision
Lost of balance	Dry mucosa membranes
Feeling of flight	Dry, hot, red skin
Staggering	Peripheral vasodilatation
Incoherent speech	Hyperthermia
Husky voice	Diminished bowel motility (± paralytic ileus)
Visual and auditory	Urinary retention
Hallucinations agitation	Tachycardia
Somnolence	Cardiogenic shock
Coma	
Central respiratory failure	
Seizures	
Death	

ing the action of acetylcholine. It is a tertiary amine which crosses into the central nervous system and reverses both central and peripheral anticholinergic actions. Physostigmine reverses toxic effects of belladonna alkaloids but must be used with caution since it can produce seizures and exaggerated parasympathetic responses. Atropine should be available to reverse its effects if necessary. Benzodiazepines and intravenous liquids may be administered concomitantly for patients suffering from serious symptoms like convulsions, delirium, coma, and hyperthermia.

The adult dose of physostigmine for severe poisoning is 2.0 mg slowly IV over 2 minutes. Too rapid IV push administration can precipitate seizures. A dose of 1 to 2 mg in adults may be repeated after 30 to 60 minutes if the symptoms reappear and then every 30 minutes to 2 hours because physostigmine's duration of action is short compared to that of the anticholinergics (5). In children, 0.02 mg/kg intravenously slowly over 2 minutes can be administered every 5-10 min at less than 0.5 mg/min, not to exceed 2 mg (4, 5). If necessary this dose may be repeated after 20 minutes (6). Pupillary dilatation produced by atropine may persist for hours or days following injection of the antidote physostigmine and thus should not be used as a guideline for therapy (7). Neostigmine has also been successfully used to reverse the toxic effects of *Atropa*

belladonna (2). However, neostigmine is a quaternary amine and does not penetrate the central nervous system well and should not be used for the central symptoms of *Atropa belladonna* intoxication.

CONCLUSIONS

A proper index of suspicion and a thorough medical history with respect to medicines, eyedrops, and berries or plants consumed may facilitate the proper diagnosis of *Atropa belladonna* or other parasympatholytic intoxication. Patients suffering from *Atropa belladonna* or other anticholinergic syndrome intoxication may first seek ophthalmologic care, so familiarity with this clinical entity is important in order to avoid unnecessary testing and provide expedient appropriate treatment when required.

The authors have no proprietary interest in this study.

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REFERENCES

1. Ceha LJ, Presperin C, Young E, Allswede M, Erickson T. Anticholinergic toxicity from nightshade berry poisoning responsive to physostigmine. *J Emerg Med* 1997; 15: 65-9.
2. Caksen H, Odabaş D, Akbayram S, et al. Deadly nightshade (*Atropa belladonna*) intoxication: an analysis of 49 children. *Hum Exp Toxicol* 2003; 22: 665-8.
3. Lee MR. Solanaceae IV. *Atropa belladonna*, deadly nightshade. *JR Coll Physicians Edinb* 2007; 37: 77-84.
4. McEvoy GK, ed. American Hospital Formulary Service Drug Information. Bethesda: American Society of Hospital Pharmacists; 1994.
5. Perry PJ, Alexander B, Ellingrod VL. Anticholinergic psychosis. *Clin Psychopharmacol Semin* 1996-1997.
6. Waternberg NM, Roth KS, Alehan FK, Epstein CE. Central anticholinergic syndrome on therapeutic doses of cyproheptadine. *Pediatrics* 1999; 1: 158-60.
7. Granacher RP, Baldessarini RJ. Physostigmine: its use in acute anticholinergic syndrome with antidepressant and antiparkinson drugs. *Arch Gen Psychiatry* 1975; 32: 375-80.